The American Journal of Surgery

EDITED BY THURSTON SCOTT WELTON, M.D., F.A.C.S.

October 1941

SYMPOSIUM

Thoracic Surgery

Edited by

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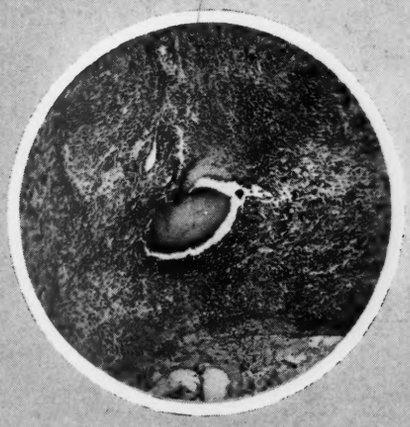

At 120 hours, histological section of size 5-0 medium chromic catgut demonstrates little cellular activity in tissue adjacent to suture.

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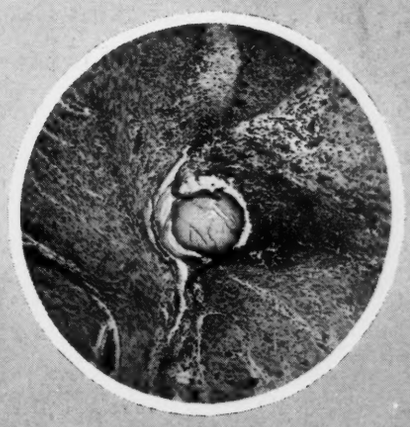

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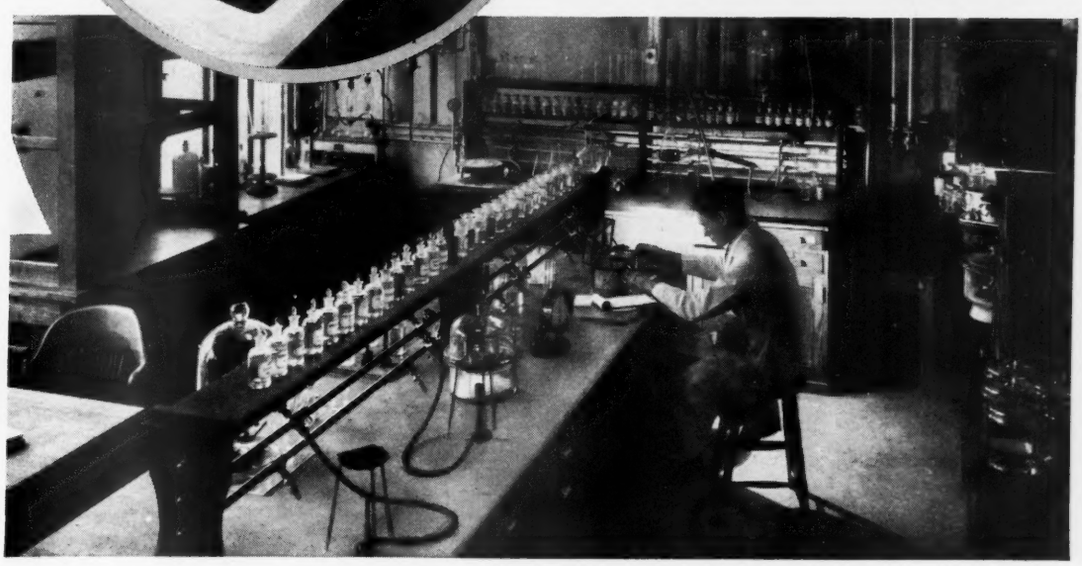

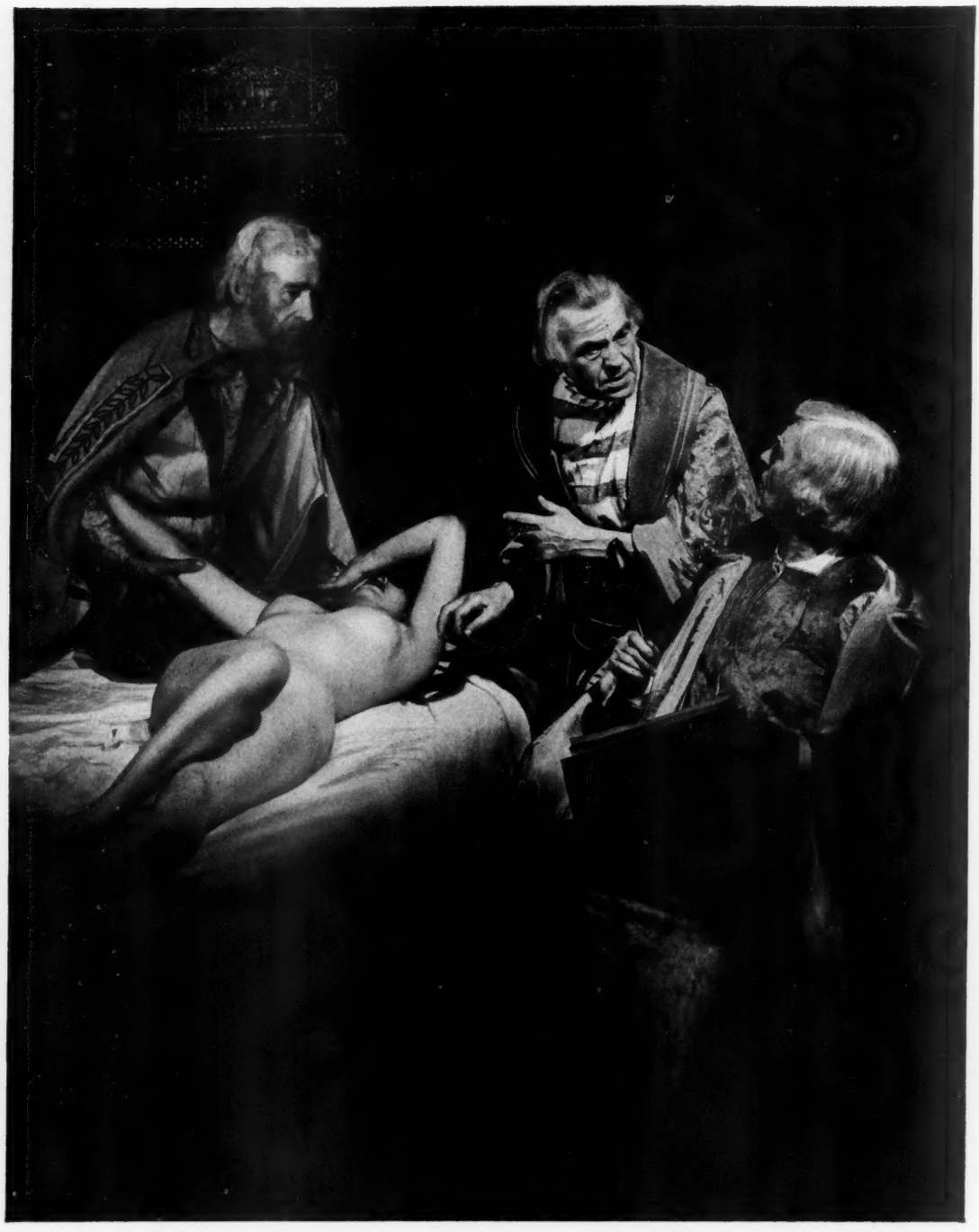

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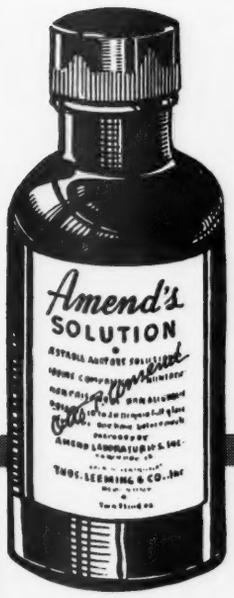

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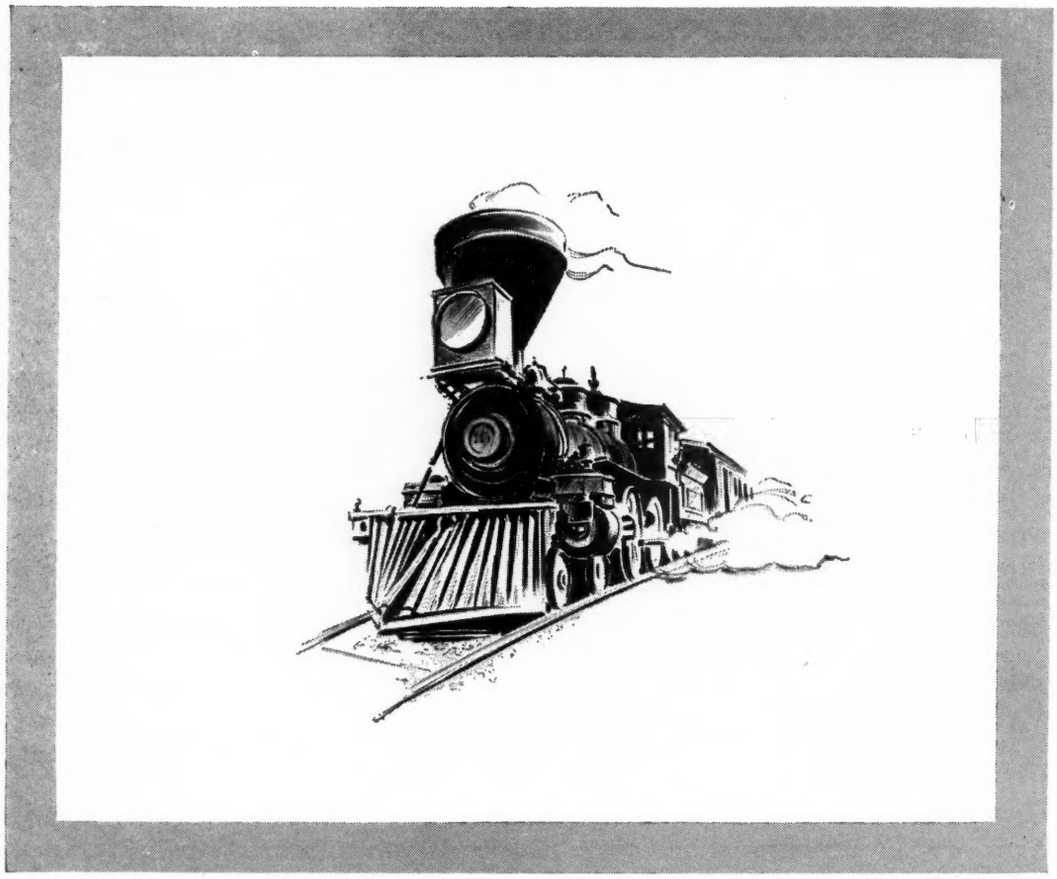

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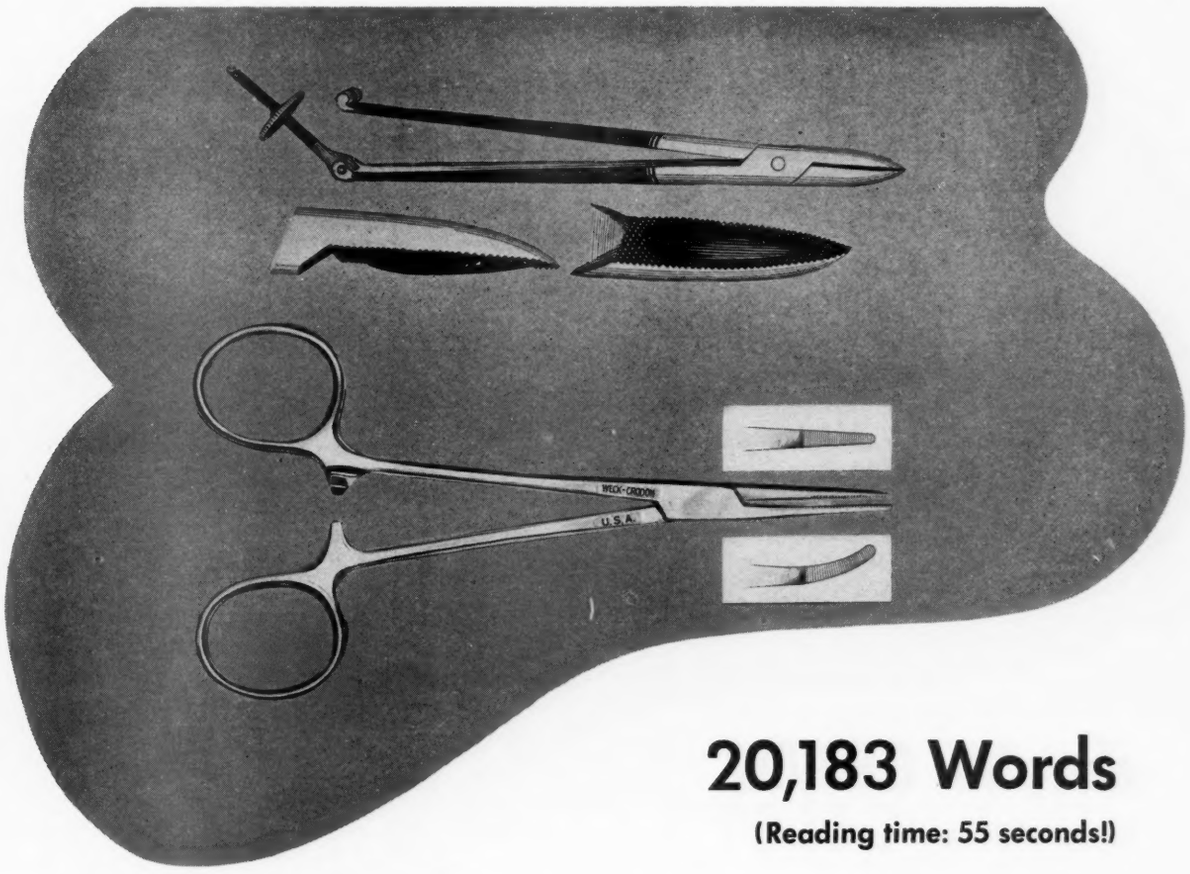

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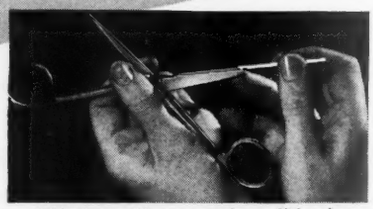

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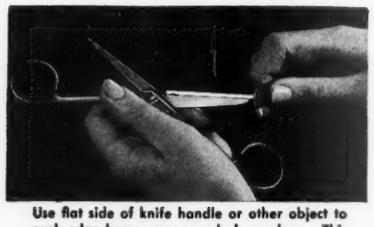

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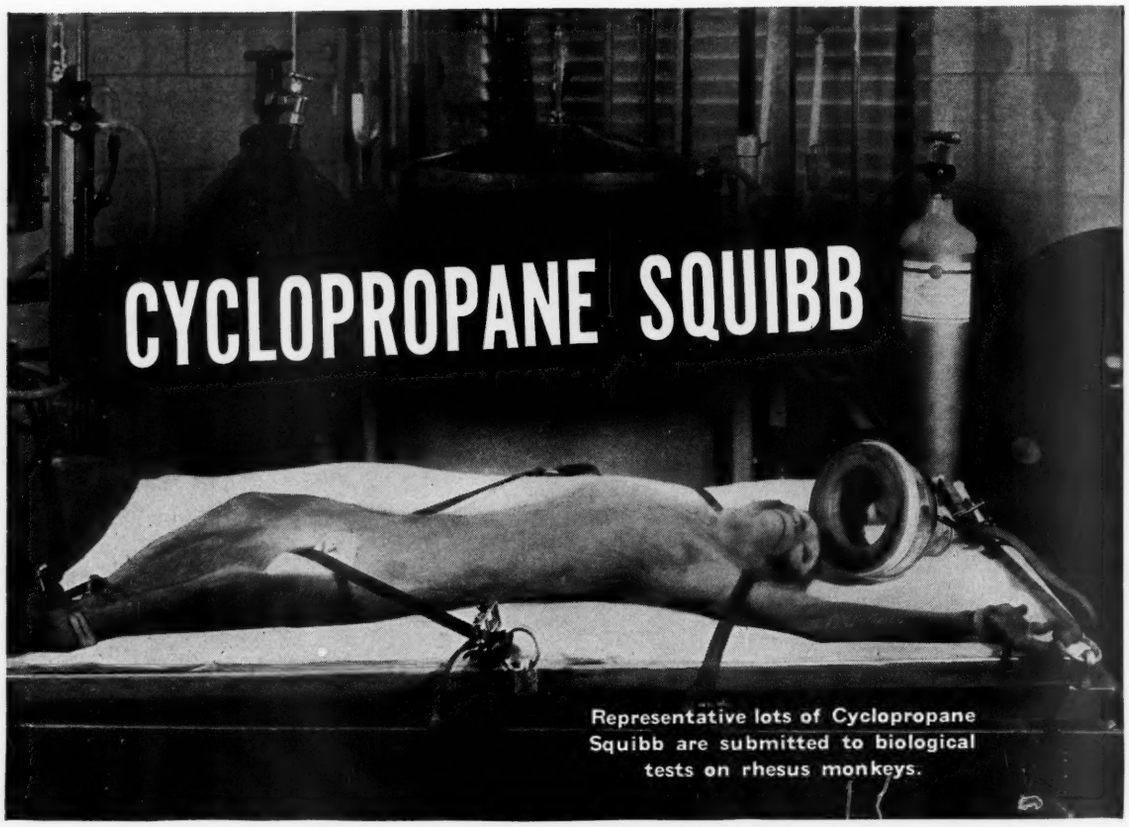
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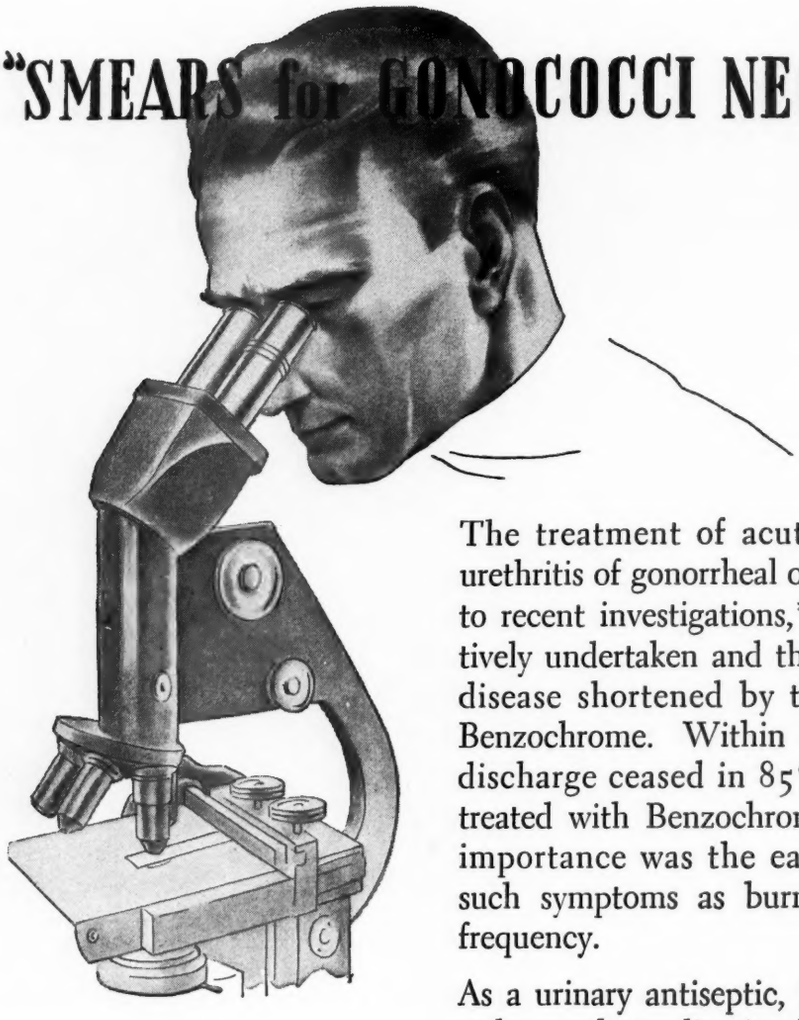

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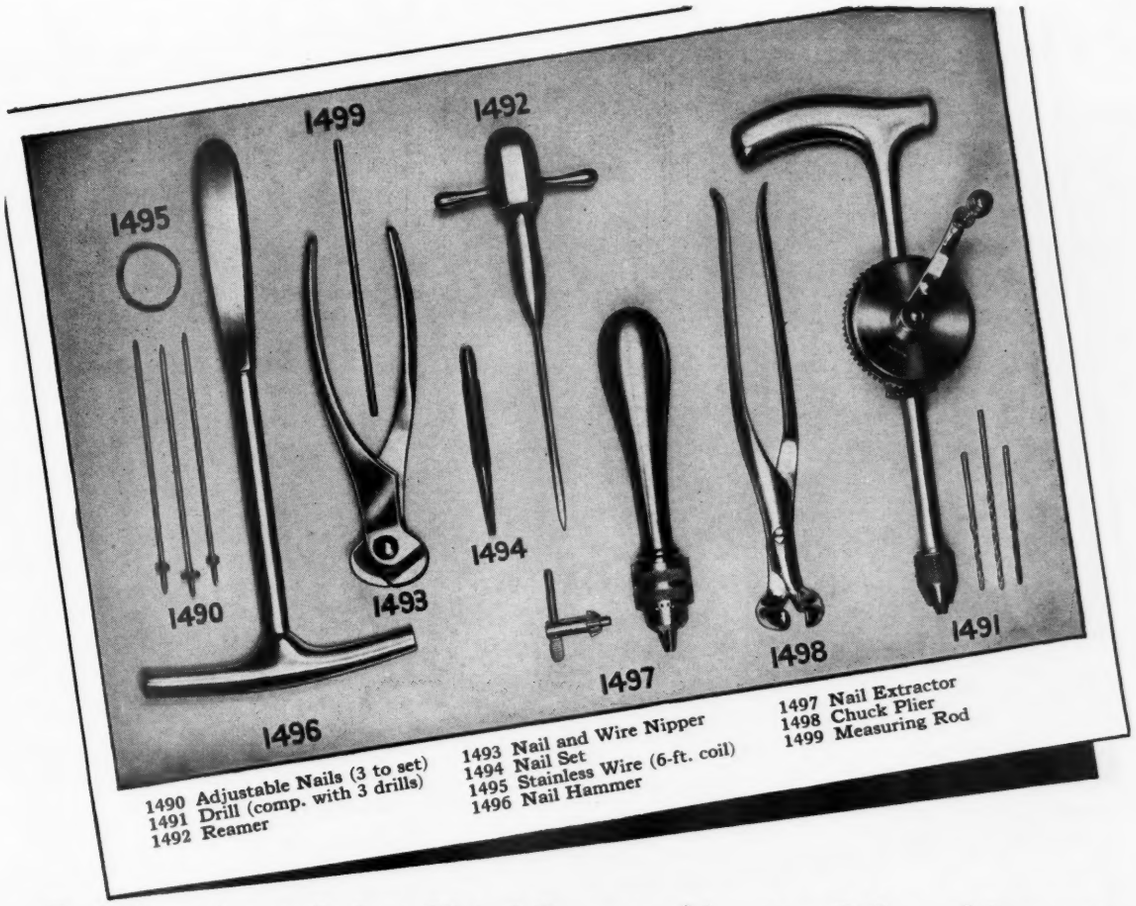

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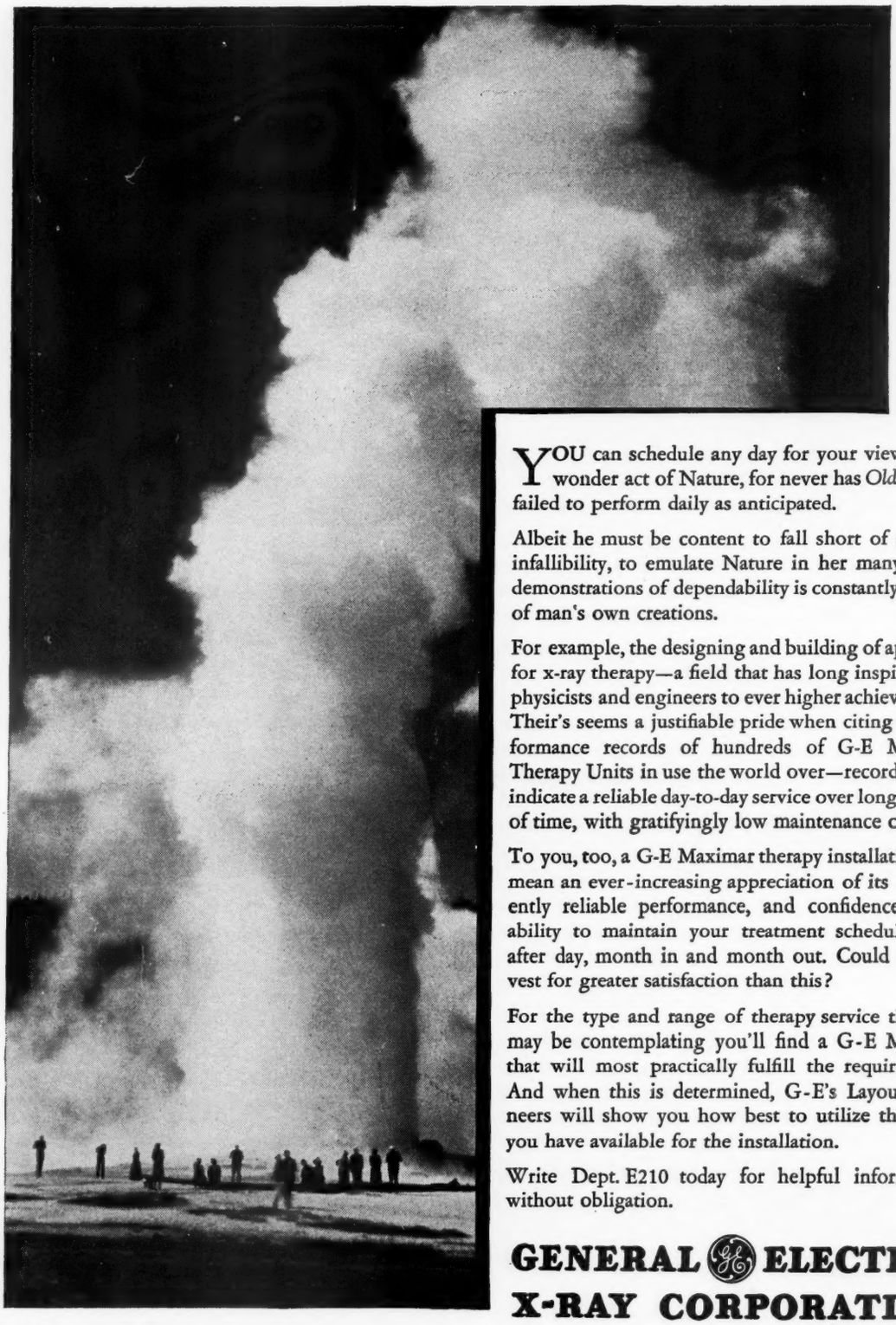

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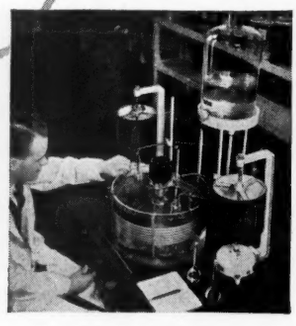

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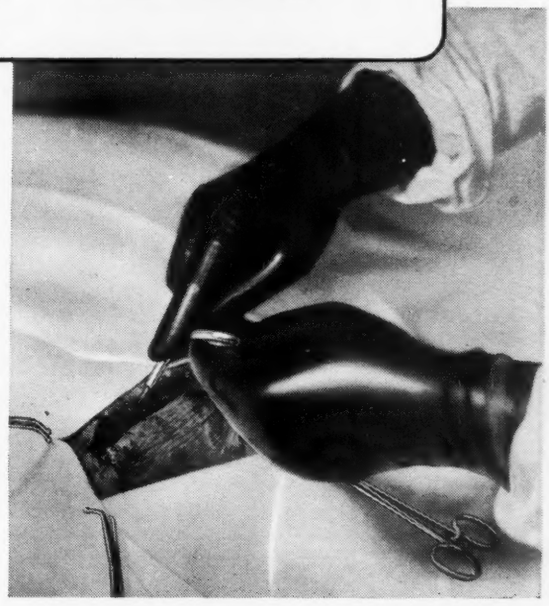

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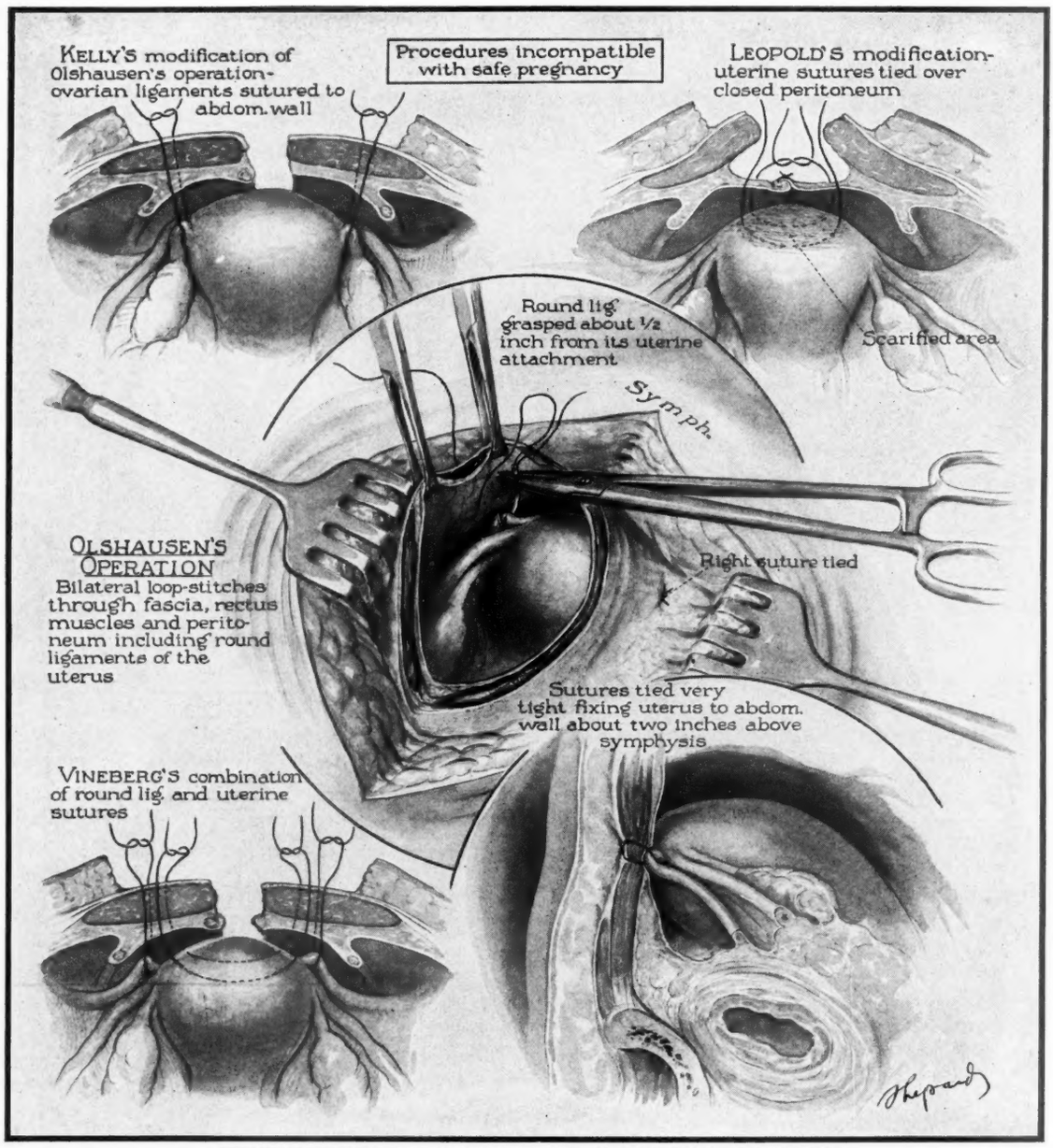
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NEW SERIES VOL. LIV OCTOBER, 1941 NUMBER ONE

Editorial

HOW MAY DOCTORS HELP MOST IN CONTROLLING PULMONARY DISEASE?

CONVINCING evidence has been presented by the contributors to this Symposium that many patients suffering from serious pulmonary disease may receive great benefits from surgical treatment. Safe surgical exploration of the thoracic cavity has permitted a correlation of clinical and pathological findings during life which have clarified many issues at stake. A more direct attack upon pulmonary disease is now possible. Drastic reduction in mortality rates for elective surgery of the chest wall, lungs, esophagus and even heart have been effected. Periods of postoperative disability can now be foretold with reasonable certainty. The degree to which health will be restored can be predicted with greater accuracy.

Although the past decade has seen distinctive and practical contributions in the field of thoracic surgery, there is reason to believe that in the near future success in handling certain pulmonary diseases will be even more noteworthy. Improvements all along the line may be expected. Perfection of technical phases, which are the immediate concern of the surgical team, will continue. However, it will be impossible to show in the future the same accelerated reduction in operative mortality and morbidity that has occurred within the past few years. For example, the mortality in partial resection of the lung has been dropped from over 20 to under 5 per cent, and in total pneumonectomy from 50 to under 10 per cent. It is therefore obvious that any great increase in the effectiveness in handling certain pulmonary diseases cannot be attributed, except in small degree, to a possible further shrinkage of the risk of operation.

The great impetus to effective handling of thoracic disease will come from the other end of the line. The profession at large, upon whom rests the responsibility for the apprehension of disease, should so improve upon the timing of their diagnostic work that therapeutic efforts will be directed to a far greater proportion of favorable lesions.

The discovery of pulmonary disease during its incipiency is a fertile field that has not been well cultivated; in fact, the profession can almost be accused of negligence. For example, let us consider two of the most important pulmonary diseases, tuberculosis and cancer. The former ranks as the number one cause of death in adolescence and early adult life, and it ranks first in economic loss. The second ranks near the top of the list of cancers which cause death, and it has become one of the most impor-

tant of all pulmonary diseases as emphasized by Blades and Overholt in this Symposium. It is appalling that the score on early discovery of tuberculosis is not greater than 20 per cent, as indicated by reports on sanatoria admissions in this country. The score on the discovery of curable pulmonary malignancy is also appallingly low—in the neighborhood of 15 per cent. By waiting to let the lesion in either case progress to the point where the doctor is certain that the patient needs an *x*-ray, thereby justifying the expense of such an examination, we simply make it difficult or impossible to obtain satisfactory results from treatment. By a simple reversal of the ratio of minimal (20 per cent) to advanced lesions (80 per cent) which come up for treatment, the whole picture in tuberculosis would change overnight. We would then promise 95 per cent good results on 80 per cent of the whole rather than limiting the high batting average to a minority group and then struggle to obtain doubtful results with the major proportion of the tuberculous population. The same applies to primary malignancy of the lung. If the majority of the lesions were uncovered in their pre-symptom or early symptom stage, the profession could boast of the highest cure rate for any internal cancer.

This is not wishful thinking as we know there is within our reach a method of bringing many pulmonary lesions to light in their early stages. This statement is true irrespective of the fact that in the case of either disease there may be no early symptom or no abnormal physical sign. It is true regardless of the fact that when symptoms and signs do appear they may be considered inconsequential, variable or unreliable. The one action the medical profession can take which will double or treble the number of early diagnoses made in chest disease is to see that all patients who come to them for any advice have a single roentgenogram of the chest at least once a year.

Doctors must all be made to realize that no matter how carefully or painstakingly the physical examination of the chest is done, absence of abnormal palpatory findings, percussion sounds or ausculatory sounds do not spell freedom from disease. Neither does absence of symptoms mean that there is no latent disease. A negative physical examination without a negative chest film gives no assurance that the patient will not be dead next year of tuberculosis or pulmonary cancer. The profession has long recommended to the public that they get in the habit of submitting to an annual physical examination including urinalysis, blood counts, and Wassermann test. To this should be added a single film of the chest. Hospitals should routinely *x*-ray all patients on admission.

Tuberculosis workers have long been convinced of the fact that if this communicable disease is ever to be controlled the population generally will have to be screened if all unsuspected cases are tracked down. Sanatoria staffs are reaching out as far as they can but their coverage is wholly inadequate. The United States Public Health Service might take over and the State carry out compulsory isolation of all positive sputum cases. Most physicians would resent such a socialized step. A practical solution can be worked out with a co-ordinated effort of existing agencies with practicing physicians and hospitals taking over the responsibility of patients under their care.

Expense has been the chief obstacle to the inclusion of chest roentgenography as a part of a physical examination. A search is well under way to perfect accurate and inexpensive ways to produce chest *x*-rays wholesale. Sensitized paper is available at about one-half the cost of the full-sized celluloid films and is almost as accurate. A method of recording the fluoroscopic image on a 35 mm. film has also been developed. Potter[1] working in collaboration with the General Electric Corporation Laboratories has developed a unit for taking a 4 by 5 inch film. This method has been found by Douglas and Birkelo[2] to be

reasonably accurate showing an error of but 2.7 per cent. Special equipment for miniature chest roentgenography may eventually be available in large *x*-ray offices and hospitals. For the moment, the initial cost of special equipment will probably limit the number of units which will find their way into general use.

For the time being, doctors must rely on existing *x*-ray facilities. Increasing use should be made of out-patient departments of city, county and state sanatoria for patients who cannot afford to pay costs. Doctors in private practice should make arrangements with roentgenologists and hospitals so that their patients may be referred for a routine chest film at a cost adjusted to make the service possible. Charges of $5.00 to $15.00 for an *x*-ray examination of the chest are prohibitive for a routine service. These prevailing fee schedules have been based upon a more time-consuming examination which includes fluoroscopy and frequently stero films. In routine work the patients are handled entirely by technicians so that the actual cost of taking and developing each film is not great. Roentgenologists can read from seventy-five to one hundred twenty-five routine films an hour. From a practical standpoint, therefore, it would seem entirely possible to bring the cost of routine single chest films within the reach of all private patients of any doctor.

It is within our power to discover pulmonary disease in many patients well in advance of the time that it becomes life threatening. Action must be taken before symptoms become so alarming that the issue is forced. By virtue of the fact that the lung lends itself so well to *x*-ray scrutiny, doctors are in a position to give real protection to their patients as far as serious pulmonary disease is concerned. The finishing touches can be put on the program to eradicate tuberculosis, and while this is being done many patients who harbor unsuspected malignancy may be saved from a certain death.

Richard H. Overholt, M.D.

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Original Articles

ANESTHETIC PROCEDURES IN THORACIC SURGERY

SIDNEY CUSHING WIGGIN, M.D.* AND PHILIP E. SCHULTZ, M.D.†
BOSTON, MASSACHUSETTS

INTRODUCTION

ANESTHESIA for thoracic surgery has been one of the prime factors in converting the hitherto formidable procedures of this branch of surgery into safe, helpful and curative measures. Vast strides have been made in the past ten to fifteen years in thoracic surgery during which we have witnessed the development of operations for pulmonary tuberculosis, and in the past eight years, removal of the whole lung for extensive bronchiectasis or tumor. In anesthesia we have seen the introduction of the endotracheal tube for the control of respirations, positive pressure and suction drainage of mucus, pus and blood. The carbon dioxide absorption technic has permitted the anesthetist to administer anesthetics with a minimum loss of body heat, less disturbance to the O_2-C°-2 ratio and has enabled him to employ the newest gas, cyclopropane, with the highest possible oxygen concentration and safety to the patient. Oxygen analysis of the blood from the pulmonary artery and vein and from the aorta have shown saturation of 95 to 100 per cent[1] under cyclopropane anesthesia. During spasm, however, the oxygen saturation dropped to 70 per cent. It has been argued in the past that thoracic surgery did not progress due to lack of these essentials in anesthesia.

We find a diversity of opinion among our leading chest clinics as to the type and anesthetics employed. Is it any wonder when one considers the short span of time covering the developments of thoracic surgery? The anesthetists and surgeons of England, Australia, Canada, Sweden, Germany and this country have been pioneering in a field of anesthesia. Only by contributions from the trials and errors of these pioneers has the present status of anesthesia in these difficult surgical procedures been developed. It is our purpose to state our experiences in over 2,000 thoracic operations of all types, describing the technics which we have found safe and which have permitted our surgeons to operate unhampered by the bug-a-boo of stormy induction, uncontrolled anoxemia, the overflowing airway and prolonged depression of reflexes.

The requirements of an ideal anesthetic for thoracic surgery have been clearly outlined by Eversole and[2] Overholt. In their experience cyclopropane fills this necessity. Beecher[3] states that ether, with nitrous oxide induction is the agent of choice in thoracic surgery because it is well tolerated by the circulation and does not depress the respirations and is nontoxic to vital tissues. Barbour,[4] Bourne,[5] and Stehle[6] find ether causes increase in blood concentration and production of acidotic states. Bourne rec-

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ommends cyclopropane-oxygen or spinal anesthesia in these handicapped patients.

In this series are included anesthesia for the various stages of thoracoplasty, extrapleural pneumothorax, internal pneumolysis, pneumonectomy, lobectomy, abscess of the lung, empyema, diaphragmatic hernia and surgery of the heart.

The patients, as a result of their pathological processes, present varying pictures of altered physical states.

The more common factors that make these patients different from other surgical patients are lowered vital capacity and vital reserve, excessive secretions of the respiratory tract, secondary anemia and sepsis. Due to their disease there is a reduction of available absorptive surface of the lungs which limits the absorption of both the anesthetic and oxygen.

Some of the complications that are created by the operation, and which make anesthesia for these cases difficult, are, rib stripping, open pleura, paradoxical breathing, stimulation of the mediastinum, traction on the lung causing vagovagal reflexes which create cough, respiratory spasm and obstruction, arrhythmias of heart and respiration; in lobectomies excessive secretion, pus, and blood and lung detritus, and posture of the patient on the operating table. In operations on the lung it is often necessary to have the patient lie on the healthy side, thus it is partially immobilized in the dependent position which limits its excursion as well as that of the diaphragm on the same side. This condition is aggravated further by the front and rear braces which hold the patient in position and the elevation of the bar or pillow which is used to widen the intercostal spaces of the operable side of the chest.

The ideal anesthetic should be: nontoxic to vital tissues, nonirritating to the respiratory tract, rapid acting for induction to eliminate stimulation of secretory reflexes and excitement, controllable for maintenance of anesthesia, with low concentration to ensure complete oxygenation, quiet respirations and prevent anoxemia. It should be a rapidly reversible anesthetic that will allow the patient to return to cough reflex and swallowing as soon as the operation is finished.

A sufficient airway should be provided with nasal tubes, oral airway, intratracheal catheter. The carbon dioxide filtration method should be employed with facilities for positive pressure and suction of secretions.

Patients are prepared with adequate bed rest, special diets and treatments. Blood examinations, vital capacity estimates, and selection of patients for surgery are done at conferences between internist and surgeon.

Many of the patients, especially those with tuberculosis and bronchiectasis, have excessive infectious secretions, very often with cavity formation. Postural drainage has aided greatly in the management of these patients. This procedure is carried out in the morning of operation. If the patient is known to have a large amount of secretion and is unable to evacuate it himself, bronchoscopy is performed preoperatively.

The ideal time of the day for chest operations is in the afternoon, so that postural drainage may be adequately carried out before hand. By so doing, it has not been necessary during the past five years to do intratracheal intubation on any thorocoplasty. To obtain sufficient drainage during operation, the operating table is placed in ten degrees Trendelenburg position before the anesthesia is started. In only a few cases has any secretion been found in the face piece after operation.

The proper posture of the patient on the operating table is one of the important factors which determines the smooth running of the operation and the anesthesia. For the surgeon it means speed of operating, good hemostasis, proper exposure of the field of operation, and the elimination of the discomforts, the presence of which leads to fatigue and oftentimes loss of equilibrium. With good posture of the patient during operation, respirations are not impeded to any great extent, with the

result that the patient is kept in a state of physiological oxygenation. Also, the control of any abnormal secretion is better facilitated.

MAINTENANCE OF AN ADEQUATE AIRWAY

Before discussion of the methods of anesthesia for thoracic operations, technics for the preservation of the air passages during anesthesia with intratracheal catheter, nasal tube, oral airway, suction of secretions from the respiratory tract and application of positive pressure will be considered.

Intratracheal intubation is used in the operations for pneumonectomy and lobectomy for four reasons: (1) to insure a patent airway for exchange of the anesthetic mixture, (2) to provide pressure when it is needed, (3) to afford an unimpeded avenue for the emergency application of Guedel's artificial respiration or controlled breathing with oxygen during apnea, and (4) to offer passage for the suction of pus and blood from the respiratory tract during operation. Whenever the intratracheal catheter is used a Water's balloon cuff is always placed one inch from its tip and inflated with 5 cc. of air after intubation. This is to insure a tightly closed system and prevent gaseous exchange around the intratracheal catheter in the trachea. No complicating infection or damage and no accident from the balloon has been noted, except that occasionally a balloon will collapse from a leak in its wall. If the balloon fails to function, a vasalined bandage is packed into the mouth about the intratracheal tube. A rolled bandage makes a convenient prop to prevent closure of the teeth on the intratracheal tube. The intratracheal intubation is carried out by one of two methods: For tumor of the lung or nonsecretive cases, the patient is induced with cyclopropane-oxygen, with or without a small amount of ether to obtain complete glottic relaxation before the intratracheal tube is introduced. In cases of cavity formation or excessive secretion as bronchiectasis, the pyroform fossae, glottis, and trachea are ansethetized with larocaine or cocaine 4 per cent and 5 cc. of 2½ per cent solution is injected into the trachea with a curved tube introducer. In fifteen minutes the intratracheal tube is inserted with an Everscope or Guedel Laryngoscope. The ease with which the tube can be introduced under local anesthesia has led to its use more and more, even in cases without secretion. During the past five years, this method has been employed almost universally. The type of catheter preferred is the silk woven, or Magill, No. 34 for women and No. 38 for men. For aspiration of secretions during operation No. 16 to 20 French catheter is passed through the intratracheal tube.

The gas machine is then connected to the intratracheal tube by means of an adapter which is inserted into the face piece holder of the rebreathing tubes and induction with cyclopropane-oxygen is performed with carbon dioxide filtration technic. Very rarely a severe spasm of the bronchi may ensue which is overcome with rhythmical positive pressure on the rebreathing bag with a very high oxygen concentration of the cyclopropane-oxygen mixture.

If the intratracheal tube is not considered necessary or contraindicated, rubber nasal tubes are used in each nostril. These are inserted as soon as the nasopharyngeal reflex is sufficiently depressed and they are retained for the duration of the operation. These tubes not only prevent pharyngeal spasm but afford an avenue of drainage for any secretion from the respiratory tract or mouth. These airways are left in place until the reflexes of the nose and throat are established in order to prevent the possibility of masseter spasm resulting in aspiration of vomitus or secretion. Also, postoperatively, they tend to stimulate nasopharyngeal and cough reflexes which aid in expelling any retained secretions in the respiratory tract and aid in stimulating the patient to a conscious state and normal control of reflexes.

POSTOPERATIVE INTRATRACHEAL SUCTION

Intratracheal suction is performed routinely immediately postoperatively by passing a No. 24 French catheter through the nose. By stimulating the glottis a cough reflex is instituted; this act opens the glottis and the catheter is passed into the trachea. If this fails, bronchoscopic suction is carried out in cases with retained secretions.

POSITIVE PRESSURE

Positive pressure is very simply performed by manual pressure on the rebreathing bag of the gas machine provided all joints of the closed system are tight. This pressure should not exceed 20 mm. of mercury pressure. The safest measure to individualize[3] positive pressure is by the direct observation of the lung by the anesthetist or surgeon to insure against overinflation. When one thoracic cavity is opened, there is no real need of positive pressure until the surgeon wishes to reinflate the lung just before closing the thoracic cavity. But if both thoracic cavities are opened intentionally or accidently, positive pressure becomes a vital necessity.

ANESTHESIA TECHNIC

Preliminary Medication. When cyclopropane-oxygen is used solely as in empyema operations, or in combination with regional novocaine anesthesia, atropine is substituted for scopolamine as it has more depressing action on the secretory glands than the vagal reflexes of respiration and circulation. The average dosages of the drugs used for preliminary medication are nembutal gr. 3, two hours before operation and morphia gr. $\frac{1}{6}$, atropine gr. $\frac{1}{150}$, one hour preoperatively. In the extremely ill patients the morphia is eliminated and nembutal is reduced to a minimum.

In preparing the patient for regional anesthesia, nembutal gr. $1\frac{1}{2}$ to gr. 3, p.o. is given two hours before surgery to which is added morphine sulfate gr. $\frac{1}{6}$ to $\frac{1}{4}$ with scopolamine gr. $\frac{1}{150}$, one hour prior to surgery. Upon arrival at the operating room, the patient's reactions to the medication are noted. Blood pressure, pulse, and respirations are checked and compared with his preoperative readings. If the patient is drousy or quiet, the paravertebral injection is then made and the reactions noted. When the effects of the medication appear insufficient or when the paravertebral injection disturbs the patient too much, intravenous morphia gr. $\frac{1}{6}$ is given. Adriani points out that intravenous morphia disappears from the circulation in one hour and clinically our experiences bear this out, for quite frequently the need for additional medication makes itself apparent and the same dose is repeated. It is to be remembered, however, that morphia given intravenously is about one-third more potent than the same dose given subcutaneously. The preparation and injection also differ from the subcutaneous method in that the dose is dissolved in 2 cc. of saline and given slowly, allowing two minutes for the injection. The usual doses employed are gr. $\frac{1}{8}$ to gr. $\frac{1}{6}$. Very rarely have we given gr. $\frac{1}{4}$. We have used this method frequently and have noted no untoward effects. Respirations in only a few instances have been mildly depressed. We do not hesitate to use it after a year of extensive trial. Occasionally, even under this régime, patients complain or become restless, but upon recovery have no memory of the procedure and undergo the next stage without any fear. We have gone into this discussion at some length, but it is our belief that the reason for local anesthesia falling into a measure of last resort in the past and its success in our hands depends primarily upon the adequate medication of the patient according to Guedel's[7] scale of metabolic activity. Tubercular patients have a definite tolerance for these drugs, perhaps because of their increased metabolic rate and common usage more than overbalances the depressant effect one would expect in such sickly individuals. Another factor which permits the use of larger and repeated doses is the fact that no depressant agent is added

TECHNIC OF ADMINISTRATION

Thoracoplasty. Two methods of anesthesia are employed for this procedure: (1) paravertebral block with 1 per cent novocaine plus infiltration in the line of incision, fascial layers and directly around the intercostals as they are exposed. Two drops of adrenalin are added to each 100 cc. of solutions, (2) the combination of cyclopropane-oxygen anesthesia with regional infiltration and paravertebral injection with novocaine, 1 per cent, without adrenalin. The latter is omitted in the presence of cyclopropane because of its sensitizing effect on the conduction mechanism of the heart.

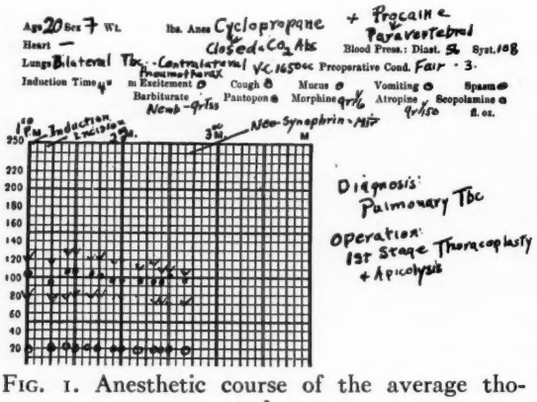

FIG. 1. Anesthetic course of the average thoracoplasty.

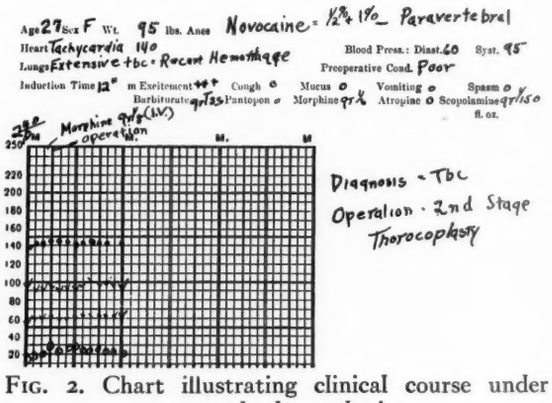

FIG. 2. Chart illustrating clinical course under paravertebral anesthesia.

Although either technic may be used for thoracoplasty, better results are obtained by individualizing each case with the selection of the anesthesia determined by careful analysis of the patient's condition. Thus vital capacity, activity of the disease, the amount of secretion, history of hemorrhage, contralateral disease or collapse procedure, physical and psychic state of the patient, history of drug sensitivity and operating time must be considered. All of these factors singly or collectively relate to (1) spread of disease and (2) operative morbidity and mortality.

As a consequence we consider those cases with (1) excessive secretions, (2) contralateral pneumothorax or thoracoplasty, (3) history of recent hemorrhage, (4) acute disease and (5) extreme debilitation, to be the type best done under the regional anesthesia while the patients in better physical state receive cyclopropane-oxygen plus regional anesthesia.

The technic for the administration of cyclopropane-oxygen anesthesia is as follows:

The bag is inflated with four liters of oxygen and the flow of oxygen is set at 1,000 cc. per minute. The patient respires this atmosphere for a few breaths and then cyclopropane is allowed to course in at the rate of 500 cc. per minute until the eyelid reflex is lost and then oxygen is reduced to 500 cc. per minute and this proportion is maintained until the eyeballs have ceased their oscillations and the respirations are smooth, equal and quiet, indicating second plane of third-stage anesthesia. When this point is reached, the cyclopropane valve is closed until the need for higher concentration of the mixture makes itself evident and the surgeon is allowed to proceed. The time of induction varies from three to fifteen minutes with the average about five minutes.

The induction with cyclopropane is followed by the regional infiltration of the line of incision and the paravertebral injection with novocaine 1 per cent without adrenalin. The paravertebral injection is made by direct vision after the exposure of the intercostal space has been made. (Figs. 1 and 2.)

TECHNIC FOR PNEUMONECTOMY AND LOBECTOMY

These two procedures although differing somewhat in their anesthesia requirements and surgical handling are grouped together for consideration because of so many points of similarity.

The preparation for both is similar in that secretions are decreased by postural drainage and bronchoscopy if necessary. Preliminary pneumothorax is established in pneumonectomy cases if possible.

The plan of premedication of the patient for anesthesia follows the general pattern described above. Our patients receive 1½ to 3 gr. of nembutal two hours before surgery and gr. ⅙ to ¼ of morphia one hour before surgery with atropine gr. 1/100 to 1/120. The patient is then brought to surgery and the throat cocainized and a cannula is inserted in a convenient vein in the ankle for fluid or blood if needed during operation. We have placed much stress on rather commonplace routine details, but it has been our experience that these procedures must be instituted before the operation is allowed to proceed.

The patient is then intubated with the technic described under the preparation of the patient. The tube and gas machine are connected and the induction with cyclopropane commences in the usual manner. The average time for the induction is about five minutes. Certain respiratory aberrations may be noted at this time, but these are usually due to either secretion, spasm or effects of pneumothorax. When respirations are under control, the patient is placed in the proper position, prepared and draped, and the infiltration of novocaine is then made.

With a tight system, little addition of cyclopropane will be necessary until the pleura is opened. The anesthetist will find it to his advantage to forestall the effects of the pleural reflex by increasing the concentration of cyclopropane a few minutes before this point is reached. When the pleura is opened, gentle positive pressure will aid in transcending this phase smoothly. Mobilization of the lung evacuates secretion from the latent cavities, and excites vagovagal reflexes. When practical, the ligation of the bronchus should be

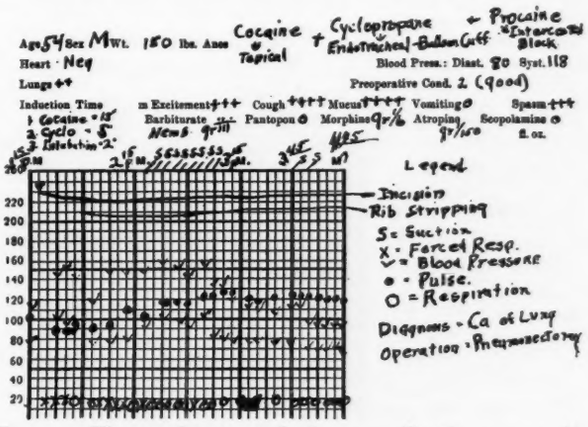

FIG. 3. Chart of some of the complications seen in pneumonectomy; marked cough reflex and secretion.

attempted as soon as possible in the wet cases for the relief of secretion and spasm, which are aggravated by continual manipulation. If spasm or cough occurs, moderate pressure on the bag will control the situation until the reflex subsides.

Just before completion of the closure of the thoracic cavity positive pressure is instituted under the surgeon's observation as to the response of the lung. The pressure is applied slowly and gradually built up. Anesthesia is maintained until the wound is closed. Bronchoscopy is then performed and all secretions aspirated. By passing oxygen through a special nipple the bronchoscope will make the transition from the anesthetic atmosphere to air less abrupt. (Fig. 3.)

TECHNIC FOR EXTRAPLEURAL PNEUMOTHORAX

Certain factors deserve consideration. First, the activity of the cough reflex from mediastinal stimulation, second, by providing quiet, even respirations, the surgeon is provided with ideal conditions for operating, and third, in the event of accidental opening of the pleura, means for application of positive pressure is essential.

We find a combination of cyclopropane inhalation and novocaine paravertebral injection with procaine the most satisfactory. Regional anesthesia alone fails to give the desired duration and does not control the cough reflex. Unsupplemented cyclopropane requires a higher concentration with consequently more depression of the patient. The technic is almost identical with that of thoracoplasty.

The procedure consists of: (1) placing the patient in the lateral thoracoplasty position; (2) inhalation with cyclopropane, using the gradual induction technic with the concentration of the gas varying between 15 per cent and 25 per cent; and (3) injection with 1½ per cent solutions of procaine paravertebrally and in the line of incision.

Lung Abscesses. There are a few cases in surgery which present more serious problems from either surgical or anesthetic angles than patients with lung abscesses. The fulminating infection produces a profound prostration of the patient, and to this are added the effects of mechanical obstruction to aeration of the lung, so that cyanosis, uncontrollable cough, with foul sputum, rapid pulse, and dyspnea complicate the choice of anesthesia.

Agents which will reduce the oxygen saturation still further cannot be used here, nor can inflammable gases or vapors be employed because of the use of cautery. Thus the anesthetist is left with the choice of regional, intravenous or rectal anesthesia. We have selected as the safest method, regional anesthesia with adequate premedication. Premedication is essential for the application of positive pressure with oxygen on the fully conscious patient from which he is apt to be more uncomfortable than from the surgical procedure. The combination of nembutal, morphia and scopolamine in judicious doses is given in the manner described previously under paravertebral anesthesia. The intravenous injection of morphia can be used to great advantage in these cases, particularly for the relief of cough.

Positive pressure with 100 per cent oxygen is helpful in these cases and is applied when the pleura is to be opened. It is used for a threefold purpose: (1) to retain the lung against the thoracic wall to prevent empyema, (2) to dam back the pus so that when the abscess is opened with the cautery the pus is forced outward instead of passing through the bronchial fistula, and (3) to afford the patient a forced oxygenation which apparently they need, as observed by improvement in color and general condition when the positive pressure is instituted. The pressure is maintained until the dressing and binder are fixed.

Procedure. The procedure is as follows: (1) medication, (2) paravertebral injection, and (3) positive pressure oxygen.

Empyema. Drainage of empyema is performed under regional anesthesia or cyclopropane-oxygen. When simple drainage is to be instituted, local infiltration is the simplest and most satisfactory. In the chronic type, however, infiltration may not be as well suited for the more extensive procedures involving resection of several ribs and thickened pleura. In these cases cyclopropane better serves the purpose.

Technic for Cardiac Surgery. Anesthesia for operations upon the heart is attended with technical difficulties for the surgeon in that he is contending with a constantly moving organ whose full activity is essential for the maintenance of the patient. Consequently, variations in pulse quality and rhythm may be expected upon handling this vital tissue and the surgeon and anesthetist must bear this in mind. The most serious complication that may occur is that of ventricular fibrillation.[8] Unless early detection with prompt therapeusis is performed, the outcome is fatal. For this reason a 10 cc. syringe is loaded with procaine,[9] ½ per cent solution and kept on the instrument table ready for immediate injection into the cardiac muscle should this complication develop.

Whether to use cyclopropane, ethylene or nitrous-oxide ether is a controversial

issue as is the necessity for intubation. Waters[10] states that there are no contraindications for the use of the cyclopropane in experienced hands. Other observers do not recommend its use in cases in which there are known disturbances in the[11] conduction mechanism of the heart. We believe, however, that these patients require an anesthetic with minimum toxicity and maximum oxygenation to spare an already embarrassed circulation. The cases which we have done with cyclopropane endotracheally have impressed us with the fact that this agent conforms with this precept.

We believe that these cases should be intubated so that a patent airway is assured at all times. Straining, spasm, cough or any interruption in the normal respiratory rhythm embarrasses both the surgeon and anesthetist. One of the early pericardectomies was done under a combination of local anesthesia and cyclopropane, but without an endotracheal tube. The patient was a robust, excitable male in the middle age group. His induction was prolonged by a moderate struggle. His respirations were rough, even with nasal and oral airways so that he was partially obstructed, yet his color was apparently unaffected. Exposure of the field was made difficult by the uneven respirations. As a result, the operating time was increased, but in this particular case, in spite of the difficulties, the patient made an uneventful recovery. Another factor worth noting was that no arrhythmias were detected under these far from ideal conditions. The contrast between the nonintubated case and the intubated cases was quite marked as far as operating conditions were concerned.

Procedure. The procedure is as follows: (1) premedication, (2) intubation, either under local or general anesthesia, (3) cyclopropane and oxygen anesthesia in 15 to 20 per cent concentrations, (3) regional anesthesia with novocain, 1 per cent.

Technic for Transpleural Approaches for Diaphragmatic Hernia and Tumors of the Lower End of the Esophagus or Fundus of the Stomach. For this procedure, we have borrowed from our experiences with other thoracic operations. The course these patients have taken has been of particular

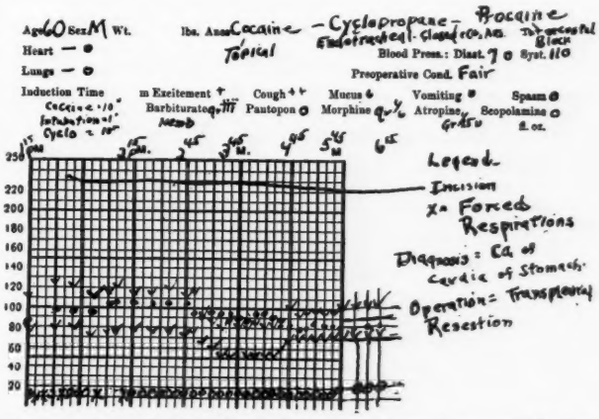

FIG. 4. Anesthesia chart for transpleural approach for resection of cardia of stomach.

interest and value. Here we are not dealing with an impaired respiratory or circulatory system, yet we must remember certain fundamental essentials. A placid field in which the activity of the diaphragm must be curtailed and billowing effect of the lung removed, can be obtained by intratracheal cyclopropane followed by injection of the phrenic nerve with novocaine when the thorax is opened. The lung collapses so that it is necessary to re-expand it at fifteen to twenty minute intervals. With this approach and technic, certain disturbing factors have been greatly ameliorated. Relaxation is no problem, for with the ribs removed and the tissues retracted, the exposure of the lower esophagus and upper stomach is obtained and maintained with facility. The ballooning bowel of the abdominal contents is absent, as are the usual complications of chest pathology. These patients seem to tolerate these procedures fairly well. (Fig. 4.)

Procedure. The procedure for the preceding technic is as follows: (1) medication, (2) Levine tube through the nose for gastric drainage is employed for stomach cases, (3) intubation under topical cocaine or larocaine, (4) regional infiltration and paravertebral block with novocaine ½ to 1 per cent, (5) gradual induction with cyclopropane in concentrations of 15 to 20 per

cent, and (6) injection of the phrenic nerve with novocaine ½ per cent.

COMPLICATION OF CYCLOPROPANE

Some of the more common complications will be considered:

1. *Cardiac Arrhythmia.* Robbins[12] concludes that arrhythmias occur with cyclopropane when morphia is added to the pentobarbital sodium and scopolamine for premedication. In this series, a group of patients were run with this technic but some still developed minor cardiac arrhythmias, therefore, morphia has not been eliminated in this series.

The arrhythmias observed have not been severe and have all disappeared before the operation was finished. It is believed that in some cases an addition of oxygen to the mixture, or shifting to nitrous-oxygen for a few minutes has caused the arrhythmia to disappear. On the other hand, the majority have vanished[13] with deepening the level of anesthesia. It has been observed, however, that with the perfected induction, namely, carrying the patient to a second plane of third stage (Guedel), before even preparing the field of operation, and combining cyclopropane-oxygen with regional novocaine anesthesia has practically eliminated arrhythmias. Another observation has been that with the reflexes under complete control (in second plane of third stage) the reflex spasm of the larynx (glottis) together with cardiac arrhythmias have been practically eliminated. A quick and more complete induction has been facilitated by substituting atropine for scopolamine in the preliminary medication when cyclopropane is used.

2. *Apnea.* As has been stated, in the patient with extensive bilateral disease, the absorptive surface of the lung is reduced both for oxygen and anesthetic. For this reason the more extensive the disease, the more difficult the administration of the anesthetic. The tidal air becomes lowered and the reflexes are more difficult to control. It is in this type of patient that complications are more apt to occur. The margin between the normal functioning respiratory center and apnea (paralysis of the respiratory center) is very narrow. As a result, the induction is usually prolonged and apnea develops very easily and very often is repeated several times during the operation. One minute the patient is phonating and crowing with laryngeal stridor and the next minute he is in complete apnea. In experienced hands this is not a serious complication. With sufficient oxygen under controlled respirations by rhythmical pressure on the re-breathing bag the patient is kept in a perfectly normal clinical course. As soon as the cyclopropane concentration in the respiratory center is lowered and the hydrogen ion concentration is raised, the patient spontaneously breathes normally, with return of normal respiratory center function. To prevent return of respiratory spasm and phonation, cyclopropane has to be added immediately. Guedel produces[13] apnea intentionally to relieve the patient of arrhythmia, or to suspend the motion of the lung in intrathoracic operations.

Hypoxia, or state of insufficient oxygen, may also develop with hypopnea, and if unrecognized and untreated may lead to apnea with further deprivation of oxygen leading to anoxia. The hypopnea may be due to the action of an overconcentration of the anesthetic on the respiratory center or a partially obstructed airway. With cyclopropane the color of the patient is no guide to the degree of suboxygenation as the patient remains pink, but the synchronization of the chest and abdomen disappears, the blood pressure becomes elevated and arrhythmia of the heart may develop. The treatment is establishment of a patent airway and adding oxygen under rhythmical positive pressure if necessary.

The possible causes of apnea are: (1) Insufficient depth of anesthesia with cyclopropane with reflex respiratory spasm; (2) narrow margin between apnea and reflex control level of anesthesia; (3) excessive carbon dioxide absorption and anesthetic

concentration; (4) excessive premedication, and (5) hypoxia.

3. *Blood Pressure Variation.* The blood pressure variations observed in chest surgery have been different from those seen in other types of operations.

In the emotionally stable, good conditioned patient, a level clinical course may be maintained in all the various types of chest operations. In the patient with high metabolic rate, and especially with debility and low vital capacity, whether given regional anesthesia and cyclopropane-oxygen combined or paravertebral anesthesia alone, the blood pressure usually rises rapidly with a corresponding drop in the course of one-half hour.

Lobectomies and pneumonectomies for bronchiectasis and lung tumor follow a remarkable normal blood pressure course with the combination of regional anesthesia and intratracheal cyclopropane-oxygen, even for operations lasting three to six hours. If there is excessive loss of blood or difficulty in the lysis of adhesions, or if extreme traction on the lung is required, a marked drop in blood pressure usually ensues.

In the first cases of the series, the few that were done with cyclopropane oxygen without regional anesthesia showed much more depression of blood pressure.

When the blood pressure falls twenty points below the patient's preoperative level, various drugs may be used. About five years ago, it was thought that coramine, 2 cc. subcutaneously, gave satisfactory results. This was replaced by ephedrine, gr. ¾, pitressin ½ cc. (Bourne[14]) which was also believed to stimulate the circulation. The results of both of these technics were very questionable as to whether the blood pressure was effected by them or the fluid therapy. In the last year neosynephrine,[15] m. 1, intravenously or m. 4 intramuscularly, has given very definite beneficial results in almost 100 per cent of these cases. If the desired results are not obtained after fifteen minutes, the same dosage is repeated.

4. *Loss of Body Fluids.* To counteract the loss of blood and body fluids and to prevent depression of the circulation, glucose in saline, 5 per cent, is administered intravenously before the operation is started after the induction of cyclopropane. In regional anesthesia local novocaine, 1 per cent, is used at the site of venopuncture. During the operation, 1,500 to 2,000 cc. of fluid is administered. If the circulation fails in spite of glucose solution, 500 cc. of donor's blood is added to the venoclysis. Patients with marked debility and those for lobectomy and pneumonectomy receive transfusions of blood for supportive or preventive measures preoperatively.

5. *Nausea and Vomiting.* Vomiting and nausea are a definite sequelae of cyclopropane anesthesia. The large majority of patients will vomit at least once on the revival of the vomiting reflex, and a small proportion have spells of nausea and vomiting for several hours; but the degree of this complication is not of the same intensity as with ether, and there is not the manifestations of acidosis or prostration with this gas as there is with ether. About 1 per cent of the patients will have spells of nausea and vomiting for twenty-four hours.

Explosions. Unfortunately, due to several fatal explosions, some medical groups have condemned cyclopropane. According to the Bureau of Mines, ether is the highest explosive of all anesthetics in the proper proportion with oxygen. Nitrous-oxide-ether mixtures have the highest explosive range.

Waters has administered cyclopropane to[16] 17,800 patients at the Wisconsin General Hospital since 1932 and Rovenstein to more than 50,000 patients in five years at the Bellevue Hospital, with no explosions. The only precaution used at these hospitals is personal contact by the anesthetist with the patient and with all objects within his reach before the anesthetic mixture is used, in order to equalize potentials of static electricity and to eliminate fire or electric current from the operating room.

The Horton megohn has been used in Boston since 1939. From a megohn attached to the gas machine four wire leads pass to the anesthetist, patient, operating table and ground. This apparatus equalizes the potentials of static electricity in all of these objects. By requiring everyone coming into the region of this hookup to contact the apparatus remote from the rebreathing circuit, all potentials of static electricity are eliminated. Other precautionary measures which should be observed are the prevention of fire or heat and electrical apparatus of any sort from coming in contact with the anesthetic mixture in the region of the rebreathing circuit of the gas apparatus. All electrical appliances with the exception of the low powered laryngoscope and extension operating lamps have been discarded. The surgeons are of the opinion that they would rather do without the electrosurgical instruments and appliances than to give up the benefits of cyclopropane to the patient.

POSTOPERATIVE CARE

At the earliest moment, usually when the surgeon is closing the chest cavity, in open thoracic operations or suturing the skin in extrapleural cases, the anesthesia is lightened by washing the patient's respiratory tract with 100 per cent oxygen under 5 to 6 mm. of mercury pressure. If this procedure is timed properly, the cough reflex and early return to consciousness may be anticipated. As soon as the dressing is applied a No. 16 to 20 gauge French catheter is inserted into the trachea through the nose for aspiration of secretions from pharynx and trachea.

By this time the patient is conscious or semiconscious. Transfer to bed is made at once and the patient is given oxygen by tent, oropharyngeal catheter, or B. L. B. Mask administration. Oxygen therapy is continued until the blood pressure, pulse, respiration and color are stabilized within a normal clinical range.

In placing the patient in bed, except in pneumolysis and extrapleural pneumothorax, the operative side is always dependent for hemostasis, to relieve pressure on the healthiest lung, to aid respiratory exchange and to support the circulation. A pillow is placed under the back to relieve pressure on the unaffected lung so that the patient lies at an angle of forty-five degrees.

Intravenous glucose 5 per cent is started in the arm at once. Dependent upon the amount of intravenous fluids the patient has received during the operation, 3,000 cc. is well tolerated for the average patient in six hours. If the patient is still in any degree of shock, a transfusion of donor's blood is run through the intravenous system.

If the blood pressure is below normal or failing, neosynephrine m. 1 is injected directly into the intravenous infusion.

The opiates are given sparingly to prevent postoperative depression of respiration and circulation. By limiting the sedation, the patient is more able to cough and raise secretions, thus decreasing the possibility of the spread of disease and atalectasis. Pantopon is preferred, as it has less depressing effect on the respirations and clinically produces less nausea and vomiting than morphia. If pantopon causes any untoward complications, dilaudid is substituted.

Table 1

Type of Cases	No. of Cases	No. of Deaths
Thoracoplasty	1939	43
Extrapleural pneumonolysis	39	
Open pneumolysis	6	
Closed pneumolysis	173	
Lobectomy	77	11
Pneumonectomy	54	7
Lung abscess	115	24
Empyema	5	
Cardiac surgery	5	
Diaphragmatic hernia	5	
Transpleural gastric resection	2	
Total	2420	85

The deaths in this series from all causes in a postoperative period of three months

have been listed. In the pneumonectomy group with the exception of the neoplasms, there has been only one death in the last twenty-eight cases, and that was due to a sulfanilamide reaction.

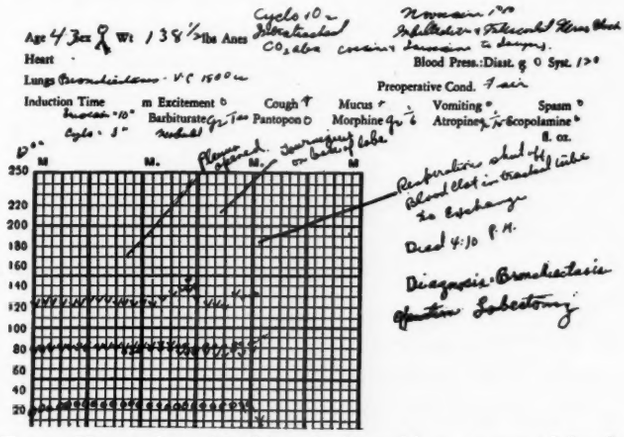

FIG. 5. Hemorrhage blocking the bronchi as a cause of death.

Deaths directly attributed to the anesthesia number six, of which none is found in the large thoracoplasty group.

Hemorrhage in the bronchus is a hazard which is difficult to overcome. In the chart below of a lobectomy, it will be seen that this accident requires but a few minutes to alter an uneventful course to that of a fatal outcome. The clot in the bifurcation of the trachea was too large and solid to be aspirated. (Fig. 5.)

Shock was the prime factor in the death of a forty-one-year old male with carcinoma of the lung, for which pneumonectomy was performed. At the conclusion of a four-hour operation, his pulse was 130 and blood pressure 90/60. Upon his return to the ward, in spite of transfusions and oxygen, he developed an irriversible state of shock and expired one hour later. An elevated blood pressure with a rapid pulse during the first hour of surgery may have been evidences of a hypoxic state from which he did not recover despite a tranquil course during the last three hours of surgery.

The other fatality in the pneumonectomy group was due to diabetic coma. The clinical chart of this twenty-four-year old, white female with pulmonary tuberculosis revealed no untoward changes. Except for nausea and vomiting in the first twenty-four hours, her reactions were normal. On the fourth postoperative day, she lapsed into coma with the characteristic picture of diabetic acidosis and expired.

The cause of death in a twenty-eight-year old, white female with bronchiectasis for which lobectomy was instituted is obscure. Although the patient was under anesthesia for seven hours, during which time there were no periods of obstruction or apnea, she showed only slight variations in blood pressure and pulse. Her cough reflex had returned for completion of the postoperative bronchoscopy. The following morning she was semicomatose and showed a paresis of the left side with a positive Babinski test. A left-sided paralysis soon followed and she expired. This case appears similar to the encephalopathy described by Courville[17] in nitrous oxide and ether anesthesia, but wherein the anoxia developed in this instance is puzzling since the common factors as obstruction and stagnation were not present.

Death occurred in a fourteen-year old boy with massive fibrosarcoma which occupied two-thirds of the thorax. At the end of a three-hour attempt to afford palliative relief, the heart stopped while dissection of the tumor from this organ was taking place. The boy ran a pulse of 140 and his respirations were shallow throughout, although his color and blood pressure were good.

A cerebral accident, whether embolic or hemorrhagic was the cause of a fatality in the lung abscess series. The patient was a sixty-five-year old, plethoric, hypertensive male who was being drained for the second time. The anesthesia was a procaine hydrochloride infiltration with positive pressure oxygen. Following cauterization of the abscess, the patient became irrational and began to struggle. Administration of oxygen was continued but the patient expired.

SUMMARY

In summary, cyclopropane-oxygen combined with regional novocaine anesthesia is employed in operations for empyema, selected thoracoplasties, extrapleural pneumothorax, pneumonectomies, lobectomies, diaphragmatic hernia, esophageal and upper stomach and cardiac surgery.

Thoracoplasties in tubercular patients in very poor physical state are performed with paravertebral novocaine 1 per cent without cyclopropane-oxygen. Local infiltration with novocaine is employed solely in internal pneumolysis and with positive pressure 100 per cent oxygen in lung abscess.

Intubation with balloon cuff catheter is carried out in pneumonectomies, lobectomies, diaphragmatic hernia, esophageal and stomach surgery when performed through transpleural approach and cardiac operations.

Positive pressure is used only when one or both pleural cavities are intentionally or accidentally opened, and for lung abscess operations.

These technics employed in the different types of thoracic surgery have approached the ideal of anesthetic procedures. They have been proved to be safe for the patient as shown by the extremely low mortality and morbidity. The benefits derived from these methods are: (1) The elimination of irritating and toxic anesthetics; (2) the prevention of hypoxia of the respiratory center and vital organs; (3) the prevention of excessive secretions; (4) they provide an ideal means of controlling excessive secretions; (5) a rapid, smooth induction is made possible in the large majority of cases; (6) control of reflexes during anesthesia is maintained with low concentration of anesthesia and a high concentration of oxygen, and (7) early return of cough reflex and consciousness is made possible.

CONCLUSIONS

Procedures of anesthesia for thoracic surgery have been presented. Preoperative treatment, care of the patient during anesthesia and postoperative management have been outlined.

Technics for various thoracic operations, the usual complications and their treatment have been described.

As a result of our experiences with over 2,000 cases, we find that cyclopropane is a safe anesthetic agent, but that its value is enhanced by the supplement of regional anesthesia. We have seen no untoward effects from routine intubation in the intrathoracic procedures, or in the operations on the heart, esophagus or stomach; on the contrary, the intratracheal tube with the inflatable cuff has been a considerable if not invaluable adjunct.

Differential pressures have been employed in lobectomies and lung abscesses without incident.

The details of six operative deaths have been portrayed. Out of these only two may be said to have contributed directly to the fatal conclusion.

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Graves' disease in children begins usually on a previously existing simple goiter or possibly seldom primarily as such. Just such children furnish the best argument for those who contend that there is a preceding simple goiter in all cases of Graves' disease, adults as well as children; for the papillations are always formed in widely distended follicles. Acute toxic goiter without eye signs is rare in children but may precede the exophthalmos by some months.

POSTOPERATIVE PULMONARY ATELECTASIS

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POSTOPERATIVE pulmonary atelectasis and the more extensive massive collapse are receiving progressively more consideration as possible complications of operative procedures. In many localities, however, they are still being considered as pneumonias and their follow-up course will certainly bear this out, since an unrelieved atelectasis will, in a great many cases, terminate as a pneumonitis or pneumonia. Too many men have the idea that the only true expression of this condition has, as its minimum lesion, a complete lobar collapse with its mediastinal shift, and fail to realize that there are minor lobular involvements which, although they do but rarely cause fatalities, contribute considerably to postoperative morbidity, and in some instances lead to pneumonia.

Postoperative atelectasis was first described by William Pasteur,[1] an Englishman, in 1908. His original article was followed by several more in which he established massive collapse of the lung as a postoperative clinical entity. It was not until 1921 that the first report of this condition was made in the American literature by Scrimger,[2] of Montreal, and in 1924 by Leopold,[3] of Philadelphia. Since that time there has been increasing knowledge and recognition of the rôle of atelectasis as a possible pulmonary complication post operatively, and it is gradually replacing the previously all-responsible pneumonia.

Considerable confusion arises in the present concept of this condition, possibly from the inconcise application of the terms and boundaries of the terms, atelectasis and collapse. They are generally used interchangeably; for consistency it would seem better to make a distinction. One can classify all lesions of the type about to be described as atelectasis, and grade them in extent as lobular, lobar,[1,8] and multilobar, depending on the extent of involvement. These terms are logical and self-explanatory; lobar and multilobar should designate involvement of the greater portion of one lobe, or of more than one lobe, whichever the case may be, with increased intrapleural negative pressure, extensive collapse of alveoli, mediastinal shift and elevated diaphragm. These correspond more or less in extent to the more popular massive collapse and are truly massive atelectasis. The lobular lesions are comparable to those in bronchopneumonia, being patchy or confluent, the findings are more indefinite and the extent of involvement less than that found in the lobar type.

In true collapse of the lung, an entity in itself, the picture is somewhat different.[4] In it, the underlying pathology is due to a decrease in intrapleural pressure (decrease in negative pressure), rather than an increase in negative pressure as is seen in atelectasis. Using artificial pneumothorax as an example, the picture is that of a complete collapse of the lung due to the introduction of air or gas (positive pressure) by some mechanism into the pleural cavity. The mediastinal shift, if any, is away from the involved lung. On *x*-ray, the collapsed area shows very little density and is only faintly visible, a decidedly different picture than that found in atelectasis. Atelectatic lung tissue contains some air; collapsed lung tissue none.[7] Atelectasis represents the changes found in the lung due to increased intrapleural pressure; collapse represents those changes due to decreased pressure. The term, "collapse," will be used henceforth in this article as denoting a pathological state of the alveoli, rather than an entity as referred to above, reserv-

ing "atelectasis" as the term to be used to include the whole group to be described.

Habliston,[5] and Coryllos and Birnbaum,[6] have made studies that confirm the fact that in atelectasis there is an increase in negative pressure in the pleural cavity of the involved lung. Normal intrathoracic pressure is said to be about minus 70 mm. of water on inspiration and minus 40 mm. on expiration. In atelectasis, the pressure is reported as ranging from minus 150 to 250 on inspiration to a minus 75 to 150 on expiration, or more. One case is reported as being as high as 442 mm. over 337.[5]

By stressing some of the more salient points in the development and course of atelectasis and emphasizing the need for early effective treatment, some attention might be brought to the value of postoperative care which is all too frequently neglected and certainly not emphasized enough in the medical literature. To many it may seem that too much stress is laid on conditions that are considered as logical sequelae to operations, but timely treatment of these so-called minor conditions will more than repay the surgeon for his effort in detecting them.

Atelectasis is not the only offending lesion in postoperative complications, but is frequent enough and important enough to deserve special note. Although no definite incidence can be determined, it is safe to say that atelectasis constitutes a considerable proportion of postoperative pulmonary complications, which in turn constitute a major portion of early postoperative pathology.

ETIOLOGY

Some writers believe that atelectasis is caused entirely by some form of bronchial obstruction, principally a mucous plug that has lodged in the bronchi; there are others who believe that this is combined with the added factor of diaphragmatic paralysis. Since the majority of lesions occur at the bases of the lungs, it is quite logical to assume that the diaphragm and also the subdiaphragmatic viscera play a part in its causation. In the recumbent position assumed by the patient both during the operation and following it, the lower lobe bronchi are not in a dependent position and should be no more prone to plugging than any other lobe, still the incidence in the lower lobes is much greater. Whether diminished excursion of the diaphragm or splinting due to pressure from below during the operation, due to reflex paralysis from trauma to the celiac plexus or due to generalized debility and surgical shock, plays a part in the development of atelectasis, is problematical, but it seems very probable. Plugging of the bronchi is present in the majority of cases, whether primarily or secondarily, and probably plays a most prominent part in the causation of lobar atelectasis, while on a smaller scale plugging of the bronchioles causes the lobular lesions. The rôle of the diaphragm, however, must not be minimized.

Working on the assumption that plugging of the bronchus from secretions or foreign bodies present there, and factors diminishing expansion of the lung are the chief causes of atelectasis, there are numerous contributing conditions that play a part. Approaching the situation from the standpoint of the bronchi, we have the chief and most popular cause in chronic bronchitis. With the already productive nature of bronchitis, there is an added incentive in the use of irritating inhalation anesthetics. Peculiarly enough, atelectasis occurs after spinal anesthesia and causes an associated hypersecretion of the bronchial mucosa. The statement is frequently made that atelectasis is as frequent after spinal or local anesthesia as it is after inhalation; this is probably true only in part. Upper abdominal operations are particularly apt to cause atelectasis with any type of anesthesia, while in nonabdominal operations the incidence is contrarily low. Rink[8] sets the percentage of chest (all types) complications after abdominal operations as 14, after nonabdominal as 1 per cent. Elkin[13] states that after upper

abdominal operation, 10 per cent of his patients developed serious pulmonary complications, while the morbidity of patients postoperatively in all cases was 3 per cent with 40 per cent of those dying. The frequency of chest complications following upper abdominal procedures is probably enhanced by the splinting and subsequent paralysis of the diaphragm from packing and retraction at the time of operation. Either at that time, or later, from temporary weakness of the diaphragm, there is collapse of the alveoli at the base of the lung and diminished aeration. A combination of factors causes secretion of the bronchi with stasis and later plugging. There appear to be two modes of development of atelectasis: One is the series of changes that follows an out-and-out plugging of a bronchus or bronchiole with subsequent absorption of air distal to the occlusion and a collapse; the other is that which follows a collapse of lung tissue from pressure below with formation of a secondary bronchial plug from stasis of bronchial secretions. In most cases, no doubt, there is a combination of these factors to form the atelectatic lesion.

Diminished aeration of the lungs predisposes to the development of atelectasis. This, together with abolition of the cough reflex, would promote stasis of material in the bronchi which could lead to plugging.

Probably a factor in addition to diaphragmatic paralysis is the splinting of the abdominal wall musculature either reflexly from underlying peritoneal irritation, or from the voluntary response of the patient to pain in the area of the incision. This could materially decrease the depth of respirations. It takes on added significance in the male, the so-called abdominal breather. In support of this is the much greater incidence of atelectasis in men than in women, 4:1. The greater incidence of bronchitis in the male also claims consideration.

Prolonged recumbent position is a factor. Postoperative distention, especially from an acutely dilated stomach may cause enough pressure to splint the diaphragm in an elevated position, thereby causing pressure on the base of the lung. There is also a definite correlation between the incidence of atelectasis and the presence of drainage for suppurative appendicitis.[9] The incidence of pulmonary complications increases with age, coincident with the increase in prevalence of bronchitis, and also of the greater reaction of a patient to pain from an operation. The injudicious use of morphine postoperatively, although beneficial for the relief of pain, in those doses suppresses respirations sufficiently to be a factor. Elkin,[13] however, advocates a sufficient amount of morphine to allay the pain of coughing.

A study would probably show that the incidence of pulmonary complications following operation is on the decline with the advent of improved methods in anesthesiology, both in the choice of anesthetic agents and the improved methods of administration. With the improvement of surgical technic, the prolonged deep anesthesia of bygone days has been largely abolished.

In institutions using the newer types of anesthesia, it is not uncommon to hear the "old-guard" of both doctors and nurses rally to the side of the old styled anesthetist with the statement that the incidence of complications is greater with the newer closed methods of anesthesia in vogue. In previous times anesthesia was given with partial anoxia and the associate increase of pulmonary carbon-dioxide tension in the alveoli; this kept the patient breathing deeply and in that way provided one of the most potent prophylactics against the development of atelectasis. Although this state may have been beneficial in one respect, it has been found to be very detrimental to body physiology.

With the apparent variability in findings, the numerous factors in the cause and development of atelectasis, and the multiplicity of contributions, it is seen that postoperative atelectasis is a complex state, and because of its relative infancy is open

to considerable controversy and leaves much to be learned by further observation.

PATHOLOGY

In the more extensive lesions with involvement of one or more lobes, the pathology is clear cut. There is a collapse of the lung tissue with a resultant shifting of the mediastinum and heart to the involved side along with elevation of the diaphragm caused by the increase of negative pressure in the pleural cavity. At first there is a collapse of the alveoli in the involved area, but after several hours, if the condition is unrelieved, the lung tissue becomes moist and edematous. After twenty-four to forty-eight hours, the process becomes irreversible, interstitial changes take place, and pneumonia and later possibly bronchiectasis or fibrosis ensue. Combined with any of these conditions, there may be a superimposed suppuration. This is true in the acute upper respiratory infections in which the aspiration of infected discharge and secretions would lead to more severe lesions and would be more apt to produce a suppurative sequel to the atelectatic state.

In the lesser lobular lesions, the picture is more variable. The areas of involvement are patchy and the presence of clinical signs depends entirely on the location of the involvement. If the areas are near the surface of the lung, findings such as diminished breath sounds, rales, etc., are in evidence. If the lesion is basilar, in all probability findings will include, in addition, elevation and diminished excursion of the diaphragm. Should the lesion be more diffuse, the findings would more closely resemble those of the lobar type. Apparently the reaction of the body with fever, tachycardia, hyperpnea and possibly dyspnea and cyanosis, is due to the rapidity of onset of the condition, for it has been noted that in the more slowly developing lesions in the chest, that is, not postoperatively with associated atelectasis, these findings are less marked and are often absent.[4]

SYMPTOMATOLOGY AND DIAGNOSIS

There are several factors that should be considered before entering into a discussion of the symptomatology of atelectasis. Some of the statements may appear quite dogmatic at first glance, but to atelectatic enthusiasts, they seem quite logical. First of all, it might be stated that in uncomplicated surgery, that is surgery in non-infected cases, and cases which have no associated infection to the condition being operated, any pathological condition occurring in the first twenty-four hours, if there is a febrile response, is in most cases the result of anesthesia and can be strongly suspected as being intrapulmonary. One of the first and most reliable signs is a sudden elevation of fever above that normally thought to be a reaction to surgery. Such diseases as urinary tract infection, upper respiratory infections and visible evidences of infection elsewhere should be eliminated as factors. A routine chest examination should be made for there might be a lesion present in addition to pathology elsewhere. In spite of the fact that no pathogonomonic signs are found in the chest, and if the fever is associated with elevation of the pulse and respirations above what would seemingly be the level for that height of fever, on probabilities, the patient should be given treatment for atelectasis. Certainly a portion of these cases will be pneumonias, or one of the less common pulmonary lesions, but a much smaller percentage than is commonly thought. Others may be the expression of an endocarditis or septicemia, the rekindling of an old infection, etc., but these will be detected in due time and no harm has been done by the type of treatment instituted for atelectasis.

Another consideration is the use of rectal temperatures. Too much emphasis cannot be placed on the value of periodic rectal temperature readings postoperatively in the suspected period. There is a good deal of inaccuracy in mouth temperatures in an operative patient who breathes partially

through his mouth either because of some dyspnea, stupor or the general lack of concentration which follows anesthesia. Rectal temperature readings give an accurate account of the febrile response of the body and in a condition such as this are invaluable.

One of the chances for error in examination of the patient lies in the postural diminution of aeration of the lung on the splinted underside when the patient is turned to be examined. Turning of the patient, however, is not to be condemned but should be condoned, for this procedure frequently aids in dislodging small plugs of mucus in the bronchial ramifications by increasing the depth of breathing in the upper lung and also placing it in a better position for drainage.

As stated before, the most suggestive finding in the postoperative atelectatic patient is the sudden rise in temperature above average in the first twenty-four hours after operation and generally in the first eight hours. Usually the rapidity of rise and the degree of rise are an indication of the extent of involvement, but this is not necessarily true. With this rise in fever, there is an acceleration of pulse out of proportion to the fever. This again is quite significant, for it gives an indication as to the amount of shift of the mediastinum and heart. Generally the pulse rate is from 120 to 140 beats per minute, but may run higher in the more severe cases. Concomitant with the rise in temperature and pulse is the rise in respiratory rate. This may be mild or severe depending on the amount of lung space compressed. Commonly in the less severe lesions, the rate becomes 28 to 30 per minute, but in the severe cases, it may rise to 50 or 60. Cyanosis of some degree is usually present; dyspnea also varies with the extent of the lesion. Dilatation of the ali nasi is often suggestive.

In examining the chest, numerous variable and often unconfirmatory signs are found. The first thing to be observed is the position of the apical impulse of the heart, as to whether there is a shift in any direction, that is, to the right or left or even upward. Minor deviations are frequently overlooked because of the normal variation in the location of the impulse, and also the indefinite fixation before operation, which, although remediable, may be sometimes justified. Typically, the lungs should show an area or areas of diminished breath sounds, depending on the extent of the involvement. If these are absent, it may frequently be assumed that the area of atelectasis is not on the surface where it can be detected. Bronchial breathing at times can be heard in areas where the obstruction is only partial and the breath sounds are conducted to the surface by the condensed collapsed lung. At times so-called atelectatic rales are present emanating from the distending alveoli adjacent to the collapse. The lung findings also may be overshadowed by moist rales that are caused by the productive bronchitis already present. Edema that has developed from an unrelieved condition may cause rales if some air is transmitted into the atelectatic area. Changes in the conduction of fremitus and of breath sounds, lag of the chest wall, dullness to percussion, etc., aid in confirming the picture. Highly suggestive is the spontaneous cough and sometimes expectoration of mucoid or mucopurulent material when the patient is turned on the uninvolved side. Some men have attempted to correlate the vital capacity of the lungs postoperatively, but there have been no conclusions as to the association with pulmonary conditions. Logically, the capacity of the lungs should be diminished according to the degree of involvement.

Because of the added expense, the use of *x*-ray is usually omitted in most private institutions, unless demanded. The typical finding is an area of density showing the involvement of the lung area with a mediastinal shift and elevation of the diaphragm depending on the degree of involvement. The appearance of a shadow on the *x*-ray is said to depend on the existence of edema

and inflammation in the collapsed portion of the lung. This reaction takes place several hours after the onset, so it is impossible to say whether the very early lesions, especially the minimal ones, would produce a shadow. In the more extensive lobar involvement there is also a narrowing of the costal interspaces.

One must not forget that atelectasis does not always occur immediately following operation. It can occur after several days and has been known to recur several times. The development of these cases depends upon the presence of material in the bronchi which on occasion plugs them and atelectasis ensues.

Atelectasis also occurs from obstructive lesions such as neoplasms, the aspiration of foreign material, etc., but these conditions are not in the scope of this article.

The duration of the atelectatic state depends on the nature and ease of eradication of the condition that prevents aeration and re-expansion of the lung. One might reasonably question the accuracy of diagnosis in lesions, which often expectorate no mucous plug, to at least give some definite evidence of the existence of atelectasis. It certainly may be assumed that in a case after treatment that shows an immediate and almost dramatic drop of the temperature, pulse and respirations to almost normal and remaining there, has been relieved of an atelectasis. In my observation, the return to normal or thereabouts of the thermal, circulatory and respiratory systems has been the criterion for relief of the condition. Chest findings are in the main unreliable. Usually, there is return of breath sounds of some degree following the removal of the bronchial obstruction, however, the remaining signs sometimes take several days to return to normal, especially if the condition is of several hours' duration and edema has developed, the so-called drowned lung.[1]

Atelectasis may be confused with all types of pulmonary lesions and differentiation is often difficult. The use of *x*-ray is beneficial in differentiation and should be resorted to when doubt arises. Confusion with pleural effusion can be allayed by the use of thorocentesis.

TREATMENT

One may anticipate to a certain extent the type of patient that will develop atelectasis. The acute processes of the respiratory tract so frequently cause severe pulmonary complications following operation, that experience has taught this to be a definite contraindication to any surgery unless it is urgent. Chronic bronchitis, bronchiectasis or any of the productive processes of the respiratory tree are prone to be troublesome. Smoking is definitely irritating in certain individuals, causing a productive bronchitis and cough. In these cases, it might be wise to suspend the use of tobacco both before and after the operation, especially before. In addition to trying to relieve these conditions as much as possible preoperatively, it is quite important to make an intelligent choice of anesthetic. Because of the irritant action of ether, it should be omitted, if possible, and a less irritating agent be substituted. This decision becomes mandatory in the more extensive lesions of the lungs. Intratracheal anesthesia is becoming more and more popular and gives the advantage of direct aspiration of the trachea and upper bronchi, both during anesthesia and immediately following operation. However, it must be remembered that a tube in the trachea is irritating and may do much to aggrevate a pre-existing affair. Premedication should be given to diminish bronchial secretions, also smaller, less suppressing doses of opiates than are commonly used.

Treatment of atelectasis is directed mainly at the release of mucus in the bronchi and re-expansion of the lung. The patient should be protected against exposure at any time and general resistance maintained by combatting shock. Proper preparation before operation is quite essential and the gentle handling of tissues during technical procedure should be emphasized. Following operation, there

are several prophylactic measures that can be taken. Rebreathing deeply of the patient with a carbon-dioxide and oxygen (5 to 10 per cent carbon-dioxide in oxygen) mixture before the patient awakens from the anesthetic, is frequently practiced. This undoubtedly re-expands areas in the lungs by promoting deep breathing. However, since the patient is not awake at that time, further co-operation in breathing deeply or in expectorating cannot as yet be obtained. After the patient has reacted from anesthesia, carbon-dioxide inhalations can be instituted at intervals of from fifteen minutes to an hour for several repeated doses. Frequent changes in position are advisable as a routine in patients predisposed to forming pulmonary complications. Although very ill, the turning may be done with care and the benefit derived therefrom.

If atelectasis is present or even suspected, more rigorous treatment should be instituted. Carbon-dioxide and oxygen inhalations are immediately given as one of the most potent treatments. The frequency and depth of respirations produced should depend on the severity of the lesion and the amount of relief obtained. In the administration of carbon-dioxide, the patient should preferably be rebreathed in a closed bag. A closed mask is applied over the nose and mouth to insure sufficient concentration of the carbon-dioxide mixture by eliminating the chance for escape of gas. The average length of rebreathing is from five to ten minutes, and should be given until a depth of respiration is reached at which the patient can no longer voluntarily withold breathing because of pain at the wound site. To promote drainage of the bronchi by placing the patient on his uninvolved side while administering carbon-dioxide is helpful. This is further improved by lowering the head in the Trendelenburg position. In this position, the involved bronchus and lung are in the upper, freely-expanding portion of the chest. Aeration of the lung is encouraged and the plugged bronchus has every opportunity to drain dependently into the trachea from where the mucus can be expectorated. There is also a possibility of bilateral involvement which should be treated by frequent changes from side to side with inhalations. Frequently, a guarded blow (manual percussion) over the involved portion of the lung, will dislodge a plug, especially when it is in optimal position for dependent drainage. An adjuvant is to support the abdomen manually to aid in coughing.[17] Jacobsen and his co-workers[6] advocate the use of artificial pneumothorax in the treatment of the more extensive cases and have shown good results. The basis of treatment consists of the introduction of positive pressure into the pleural cavity of the involved side, to overcome the increased negative pressure caused by the atelectasis and thereby restore the mediastinal balance. Also, by the introduction of pressure, the bronchus is aided in forcing out the obstructing agent. This type of treatment contests that of bronchoscopic drainage as advocated by Jackson,[11] Overholt et al.,[12] and others who show equally satisfactory results. Both methods of treatment are proficient in expert hands and when done under optimum conditions. Ordinarily, they cannot be used as a routine, and should be reserved for the more severe cases. A concerted effort should be made to relieve the condition by medical means before resorting to an operative procedure.

In place of the hypnotic and more or less vegetative state of a patient that is kept under heavy doses of morphine, the patient should be kept as little suppressed as possible. Considerable relief can be obtained by the careful administration of sedatives and hypnotics without the accompanying marked suppression of respiration. Only morphine will suffice in some cases, but the routine use of opiates in large doses postoperatively should be abandoned. Encouraged deep breathing is advised as a routine. The use of tight dressings and abdominal binders have created considerable comment as to their effect on the patient. A binder will by compres-

sion necessarily diminish the depth of excursion of the diaphragm and lower chest, and consequently diminish aeration of the lung. On the other hand, it will give the patient both a sense of support and also actual support so that he will be more willing to cough and try to expectorate mucus from the bronchi and trachea. An expectorant should be given, either by mouth, or intravenously (as sodium iodide) if the oral route is not practicable. Steam inhalations also aid in loosening secretions. By thinning and thereby loosening secretions in the bronchi, a patient is quite apt to expectorate occluding mucous plugs. Later plugging of the bronchus may sometimes be prevented in patients that have considerable bronchial secretions by giving them postural drainage in the Trendelenburg position for several hours or for shorter periods at intervals. This facilitates drainage of the bronchi and makes it much easier for the patient to expectorate mucus than in the sitting position as is frequently thought.

Routine inhalations of oxygen, unless cyanosis, dyspnea or tachycardia are extreme, should be discouraged. The increased tension of oxygen in the alveoli causes a diminution in both the depth and rapidity of respirations, and thereby would tend to preserve an atelectasis rather than overcome it. The use of nasal oxygen gives the advantage of switching to carbon-dioxide for brief periods with facility. The use of the tent eliminates this possibility because of the inability to add carbon-dioxide to the oxygen mixture in sufficient concentration to give the desired effect without overventilation and possible alkalosis.

In a recent symposium, writers at the Mayo Clinic advocate the use of high concentrations of oxygen routinely in post-operative care, stating that it assists in clearing mucus in the bronchus,[18] and also increases the concentration of oxygen to the atelectatic area, if present, and thereby will help prevent infection and edema.[19] In my experience, relief can usually be obtained by measures directed at re-expanding the collapsed lung tissue, and this is best done by the administration of carbon-dioxide mixture directly in intermittent and concentrated doses; oxygen is not used as a routine. In the minor cases, the simple expedient of rebreathing in a closed sack is worth while, the effect being derived from the accumulation of carbon-dioxide breathed by the patient.

In summarizing the treatment of atelectasis, it might be said that the most important contribution is the knowledge of detecting an early lesion before the plug has engaged in the bronchus and irreversible anatomicopathologic changes have taken place in the collapsed lung tissue. One may be guided in the type of treatment by the apparent severity of the condition. Measures such as frequent changes in position of the patient, diminished use of opiates and encouragement of deep breathing can be used as routine postoperative care, especially in those that are potential cases of atelectasis. If atelectasis is known to be present, more drastic measures such as posturing, carbon-dioxide inhalations or any other attempt to dislodge a bronchial plug and aerate the lung may be instituted. Bronchoscopic drainage or artificial pneumothorax should ordinarily be reserved for the severe, intractable cases unless facilities are optimal for that type of treatment, as is the case in some clinics. Whether the atelectasis is due supposedly to a bronchial plugging or is due to a paralysis of the diaphragm, the treatment should be the same and every effort made to relieve the condition early before further changes can occur.

After the atelectasis has been relieved, an attempt should be made to aid the patient in expectorating the secretions that usually form in the bronchi. The remainder of treatment is supportive. A mild fever may persist for several days as a result of the mild pneumonic congestion associated with the atelectasis.

CASE REPORTS

A résumé of cases that follow, will serve to illustrate some of the less common and less acceptible statements that were made in the foregoing article. Only the pertinent points will be mentioned.

Case I. This is a case of a middle-aged female, on whom an appendectomy was done for an acute suppurative appendicitis, with drainage. By the time eight hours had elapsed postoperatively, the patient's temperature had risen abruptly to 104.2°C., with an associated exaggerated rise of pulse and respiratory rates. Examination of the lungs revealed only moist rales through both lower lobe bases. There was a slight suppression of breath sounds but they were not completely absent. There was no noticeable heart shift. Cyanosis of a mild degree and some dyspnea were present. More on the suggestion of the temperature, pulse and respiration curves than on physical findings, the patient was given the routine treatment for atelectasis and in the following eight hours, the temperature, pulse and respirations dropped almost to normal and remained there. A subsequent mild fever from a pelvic peritonitis showed very little change in pulse and respiratory rates in marked contrast to the previous response. The extent of the lesion could not be determined but the response to treatment was typical of atelectasis.

Case II. An elderly male, who was suffering from a quite severe bronchiectasis, had a cholecystectomy done. Every precaution was taken in the preparation of the patient and in the choice of a mild anesthetic. Following operation, the patient was given medicated steam inhalations to promote and loosen bronchial secretions, and carbon-dioxide inhalations were given as a prophylaxis against atelectasis. After two days, the carbon-dioxide inhalations were discontinued because the patient was progressing well. Following this, the temperature rose abruptly to 102°C. with a rise in pulse and respiration. Carbon-dioxide inhalations were resumed following which the temperature again dropped. Upon discontinuing the inhalations, the temperature again rose to about 102°C. Examination of the chest at this time revealed a patchy area of bronchial breathing in addition to the diffuse areas of coarse rales that were present before, and it was believed that the patient had an area of pneumonic consolidation. This case illustrates the repeated plugging of a bronchus or bronchi from a pre-existing productive state, with probable silent plugging of one of the smaller bronchi and ensuing pneumonia.

Case III. A young male adult, who was operated upon for an acute catarrhal appendicitis with no drainage, seemed to have a slight bronchitis with cough following operation, so steam inhalations were administered. On the third postoperative day, the doctor was called because the patient was very dyspneic, and had difficulty in breathing. The temperature had shown a sudden rise to 102.4°C., together with a rise in pulse to 130. The patient appeared quite cyanotic. Examination showed numerous rhonchi and further evidence of considerable mucus in the bronchial tree. In addition there was a suppression of breath sounds throughout the middle lobe of the lung and a shifting of the heart apex to the right. The patient was turned on his uninvolved side and upon doing so, felt a sudden choking sensation which was followed by a severe coughing spell from which he expectorated a large amount of mucopurulent material. Even after this, the temperature continued to rise to 104.6°C. Because of the failure of mild treatment previously, the patient was treated more drastically with postural drainage and adequate administration of carbon-dioxide inhalations. Following this, the temperature, pulse and respirations dropped dramatically to near normal and remained there. This illustrates a delayed plugging due to an abundance of mucus in the bronchi, and also the importance of adequate treatment.

Case IV. A young female, who was operated upon for a suppurative appendicitis, with drainage, developed a mild peritonitis following operation, with intestinal distention and temperature of 100 to 101.4°C. On the fourth postoperative day, the temperature rose abruptly from 100 to 102.4°C., with the pulse and respiration rise typical of atelectasis. Examination of the chest showed a fixing of the left lung base with suppression of breath sounds and dullness to percussion. Occasional atelectatic rales were heard throughout the basilar area. The abdomen showed some generalized distention but most of the distention seemed to be over the stomach area. To relieve the gastric distention, a lavage was done with liberation of a large amount of gas. The patient was turned on the right side and rebreathed with carbon dioxide. Following this, the temperature, pulse and respiration dropped immediately

within the next four hours, and examination of the lung showed remobilization and aeration.

Case v. This embraces repeated cases of male workers with bronchitis, who developed a low-grade fever following operation with no particular rise of pulse and respirations. Without treatment, the temperature would average between 101 and 102°c. for five to six days and then return to normal by lysis. Examination of the lungs usually revealed a few moist rales at the bases. An expectorant was given to these patients routinely, and it was found that the temperature would drop within the following twenty-four hours in most of the cases, provided they had no other cause for the fever. In view of the bronchitis that was present, and the persisting fever, these cases were considered as those of lobular atelectasis—a manifestation, so to speak, of the bronchitis. One could believe that the fever was caused by the bronchitis, but it seems more probable that the productive bronchitis caused multiple plugging of the bronchioles and ensuing patchy atelectatic areas, which were relieved by the thinning of the secretions and inducement to cough. None of these patients had upper abdominal operations.

Case vi. This was the case of a middle-aged female, operated upon for an ectopic pregnancy. Following operation, the abdominal wall was quite rigid. The temperature was elevated, and the pulse was ranging from 150 to 160, with respirations between 30 to 40. Examination of the chest showed suppression of breath sounds at the right base, with dullness and lag of the chest wall. The shifting of the heart apex to that side was questionable. Because of the possibility of obtaining some relief, the patient was turned on the left side and carbon-dioxide inhalations repeated at intervals. After several of these, the breath sounds had partially returned to the right lung base, and the pulse had dropped to 120, the respirations to 26. The patient volunteered that she felt much relieved. The temperature remained elevated, however, apparently due to intra-abdominal conditions. This case illustrates the relief obtained by improving lung physiology simply by aeration.

SUMMARY

1. Atelectasis has been definitely established as being one of the commoner of postoperative complications. When one considers the immediate postoperative pulmonary complications, it apparently is the most important and most frequent condition encountered.

2. Atelectasis may follow a plugging of one or more of the bronchi or bronchioles, or may be caused directly by compression from adjacent structures with occasional secondary plugging by mucus forming from hypersecretion of the immobilized lung tissue.

3. Atelectasis may be classified as lobular, lobar or multilobar, depending on the extent of involvement, the latter two being classed as massive atelectasis in place of the misleading term, massive collapse.

4. A sudden elevation of temperature in the first twenty-four hours of the postoperative period, together with an exaggerated rise in pulse and respiration, should immediately suggest atelectasis, and steps should be taken to establish the diagnosis.

5. Because of the frequency of atelectasis following upper abdominal surgery, and following inhalation anesthesia on patients with productive processes in the lungs, these should be considered as potential cases and postoperative prophylactic measures be instituted immediately.

6. Treatment consists of thinning the bronchial secretions, assisting in expectoration by posturing, coughing, etc., and re-expansion of the collapsed lung by promoting deep respirations.

7. In addition to the typical massive atelectasis, there are minor lobular involvements which show no typical picture but do produce a postoperative morbidity.

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The nontoxic colloid goiters of the adult, . . . whether uniform or bosselated, are separable by an indefinite borderline from the next stages, either the toxic, the so-called secondary toxic (the basedowified goiters of Kocher, the toxic adenoma of Plummer), or in the degenerated colloid group marked by constitutional disturbances and cardiac degeneration.

THE MANAGEMENT OF ACUTE EMPYEMA IN CHILDREN*

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THE standardization of the treatment of any surgical condition will be effective only if certain principles are constantly borne in mind. If any standardized form of treatment is applied to every case of a disease whose course depends on so many variable factors as does empyema, poor results will inevitably follow.

The important variable factors in acute empyema are: the age of the patient, the virulence of the infecting organism and the stage of the causative initial disease—pneumonia—at the time the complicating empyema develops. It is pertinent here to stress the fact that statistical evidence of the merits of any one method of treatment of empyema is of little value unless a sufficiently large number of cases are considered, and particularly unless there has elapsed a long enough period of time during which that treatment has been used. The incidence of empyema varies greatly from year to year, as does the virulence of the strain of the predominant infecting organism.

It is too soon to state with assurance what the influence of chemotherapy on the incidence of empyema will be. There appears, however, to be a very hopeful lessening of this complication of pneumonia especially if adequate chemotherapy has been used at the onset of the pneumonia.

We can, however, report encouraging figures in the treatment of this complication of pneumonia. From 1919 through 1938 there were 467 cases of empyema treated at the Children's Hospital. Only six of these patients received chemotherapy. Those receiving chemotherapy since January 1, 1939, will be discussed later. Table 1* shows the comparative figures for the four five-year periods, and also the figures for the patients under two years of age. The gratifying drop in mortality in the last five-year period may possibly in some degree have been due to a lesser virulence of the organisms during those five years. The greater credit, however, must go to a more efficient handling of each case. A sufficiently large number of cases over a five-year period should minimize the factor of a lesser virulence of the organism from year to year. Such is not the case in a series reported for a single year. (Table 1.)

TABLE 1

RESULTS OF TREATMENT OF 467 CASES OF EMPYEMA IN CHILDREN JANUARY 1919 TO JANUARY 1939 BEORE CHEMOTHERAPY

Five-Year Period	All Cases			Patients Two Years or Under		
	No. of Cases	No. of Deaths	Mortality Per Cent	No. of Cases	No. of Deaths	Mortality Per Cent
1919–1924	94	13	14	20	7	35
1924–1929	86	11	13	21	7	33
1929–1934	150	20	13	57	12	21
1934–1939	137	3	2	46	2	4

To apply indiscriminately one form of treatment to every case of empyema regardless of the stage of the disease, the infecting organism and the age of the patient is to invite disaster. Each case must be treated as the requirements of the individual case indicate. It is to be emphasized particularly that empyema is a complication of pneumonia and must so be regarded. Empyema itself is seldom the

* Lanman and Heyl. Empyema in Children. *New England J. M.*, 221: 1003–1007, 1939.

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cause of death; it is seldom a surgical emergency. Patients die with empyema rather than because of it.

In our series just referred to the predominant organism was the pneumococcus in 80 per cent, the streptococcus and staphylococcus in about 10 per cent each, and other organisms less than 1 per cent. The cases of streptococcal infection, while small in number, had a mortality of 33 per cent, the staphylococcal group 11 per cent and the pneumococcal group about 3 per cent.

FORMS OF TREATMENT

In general there are three forms of treatment: aspiration, intercostal closed drainage and open thoracotomy with or without rib resection. It cannot be emphasized too strongly that each of these has its sphere of usefulness, its limitations and its contraindications. No one of these three or even a combination of one or two can or should be routinely used. Each case must be treated by a method or combination of methods suitable for that particular case.

Aspiration. This form of treatment is most useful in the early stages of empyema. At this time the fluid is thin, whatever the infecting organism. Aspiration establishes the diagnosis and permits identification of the organism. It is the method of choice particularly during the stage of active pneumonia. Unless the fluid is large enough in amount to give mechanical embarrassment to the patient as shown by displacement of the heart, little more than a diagnostic tap need usually be done. The presence of pleural effusion at this stage of the disease, if it is not causing mechanical embarrassment, is of relatively slight importance to the welfare of the patient. It is only one manifestation of the pneumonia. With proper infiltration of the skin by novocain, withdrawal of this thin fluid can be done with a minimum of discomfort and without moving the patient from bed. Aspiration, then, in addition to identifying the organism, provides an easy and nonshocking method of relieving the mechanical embarrassment caused by a pleural effusion particularly in those patients who are still critically ill with pneumonia.

Intercostal Closed Drainage. This is most useful in cases in which the accumulation of fluid is so great and so rapid that frequent tapping fails to give adequate relief of the mechanical embarrassment caused by the fluid. By providing continuous drainage it obviates the discomforts, small as they may be, of repeated aspiration. The procedure may be done with little less added pain and without moving the patient from bed. Often, particularly in cases caused by the streptococcus, it results in cure.

The simpler the technic used the better if certain principles are observed. We use a soft rubber catheter which is inserted through a cannula. A small area in the skin and the chest wall muscles is infiltrated with novocain. If a small incision is made in the anethetized skin with a knife, it lessens the discomfort of passing the trochar and cannula into the pleural cavity. The catheter should fit the wound snugly after the cannula has been withdrawn. The usual precautions of not allowing air to enter the pleural cavity should be observed, and the catheter should extend only a short distance, one to two inches, within the pleural cavity. After removal of the fluid, and this should not be done too rapidly, the catheter is connected with another length of rubber tubing, the distal end of which is placed under water in a container at a lower level than that of the patient's chest.

Dependent drainage is a well recognized surgical principle but it must be remembered that if there is a large pleural effusion the diaphragm is often pushed down. In such a case, if intercostal drainage is established at the most "dependent" interspace, say the ninth, the diaphragm rises as the fluid drains off and may often press the catheter against the chest wall and so interfere with adequate drainage. The seventh interspace is usually the interspace of choice for intercostal drainage. In

cases that have had intercostal drainage and later require rib resection it is important to realize that the diaphragm may be higher than at the time of the intercostal drainage. The rib above the site of intercostal drainage should always be used. This gives better drainage and will obviate the chance of inadvertently going through the diaphragm. For intercostal and for rib resection drainage, the posterior axillary line is the site of choice in the great majority of cases.

There has been a great deal of discussion about "airtight" closed drainage, tidal irrigation and so forth in the treatment of empyema. We are convinced after thorough trial that attempts to maintain "airtight" drainage by elaborate mechanical means are ineffectual after a few days. While it is desirable to have closed drainage in a sick patient, we believe there is no form of apparatus that will stay "airtight" for more than a week or two. Also the necessity for airtight drainage for a longer period than two weeks is seldom if ever sufficiently important to be of any consequence.

Tidal irrigation and similar complicated forms of drainage have been abandoned here. The simpler the form of apparatus the better. As far as irrigation is concerned, attention is directed toward preventing the catheter from becoming clogged.

If, after a week or two, particularly in the pneumococcal infections, the intercostal drainage tube is not providing adequate drainage, the condition of the patient should by that time be sufficiently improved to warrant open surgical drainage. As we have stated previously the value of intercostal closed drainage is particularly great in early cases and in young patients. Much of the condemnation of drainage by rib resection has been wrongly attributed to the operation rather than to the choice of case in which it was used. Primary open operation with or without rib resection should never be employed during the synpneumonic stage regardless of the age of the patient and seldom if ever should it be employed for patients under two years of age. It is to be noted that "primary" open operation means open operation *not* preceded by some form of closed drainage. We wish, however, to emphasize that in many cases too long continued attempts to obtain adequate drainage by closed methods should be abandoned and open drainage substituted. Enough patients have come to the Children's Hospital with chronic empyema after weeks and even months of inadequate drainage of the pleural cavity by intercostal tube to convince us that such drainage should not be prolonged much over two or three weeks unless there is obvious evidence of improvement as shown by the clinical condition; decrease in the size of the cavity and re-expansion of the involved lung. The patient whose general condition has not improved sufficiently to warrant open drainage after two or three weeks of closed drainage is a rarity, and in such cases there is usually some other complicating factor, frequently a bronchopleural fistula.

Open Drainage. In properly selected cases this is an efficient and safe form of treatment. Its chief danger is the unwise selection of cases for its use particularly as regards the stage of the disease when drainage is instituted, and the age and general condition of the patient. In private practice the usefulness of this method is limited largely to cases that have not responded well to intercostal drainage. It is important to use this method of more efficient open drainage in such cases, however, to minimize the chances of a chronic empyema. Each case must be judged carefully on its merits. If after two or three weeks of intercostal drainage there is not a satisfactory improvement, adequate open drainage should not be unduly postponed. This is particularly true in the pneumococcal infections.

Primary rib resection is seldom if ever employed in private practice or in cases under observation on the wards from the onset of the pneumonia. Paradoxically,

however, in our series the large group of patients on the wards who received a primary rib resection did much better and had a shorter hospital stay than did those receiving other forms of drainage. In the past ten years 168 patients out of 287 received a primary rib resection. The average duration of postoperative drainage for this group was twenty-four days; all ended in complete recovery. The average duration of postoperative drainage was forty days for the group treated by intercostal drainage, and fifty-seven days for the group receiving intercostal drainage followed by rib resection.

The reason is obvious. This large group of patients was referred to the hospital when they were well over their pneumonia, if indeed the pneumonia had been recognized. The child often gave a history of a "cold" with "congestion of the lungs." He was confined to bed for a period of time. The fever subsided after a week or so and re-appeared a few days later. He received little or no treatment for his pneumonia, but because of failure of early diagnosis of fluid in the chest, the necessity for surgery was realized at more nearly an optimum time than is true of patients in a better economic status. This sort of medical treatment of pneumonia is certainly not advocated. It confirms the opinion, however, that the surgical treatment of acute empyema is seldom a surgical emergency.

There was only one death in the last five years following primary rib resection and that was in a child one year of age. Our rule is now to do no primary rib resection in a patient under two years of age. All primary rib resections are preceded by a diagnostic tap. The usefulness and indication for primary rib resection are limited largely to the class of patients in the economic bracket referred to above. It is seldom if ever to be advocated in private practice. Rib resection is, however, to be used in cases that do not do well with intercostal drainage and it often is not only useful but necessary, particularly in the pneumococcal cases.

TECHNIC OF RIB RESECTION

Here again simplicity is desirable. While the site of drainage depends on the location of the pus, the posterior axillary line is the usual site and the seventh rib the usual and best location for the resection. About one to two inches of the rib are resected subperiosteally and a double flanged empyema button inserted. The lung should be freed, as free respiratory movements promote drainage, and re-expansion of the lung obliterates the cavity. Reasonable activity by the child as well as the use of blow bottles favor re-expansion of the lung. We allow and encourage them to be up and about as soon as the fever subsides. The button is seldom left in place for more than two weeks.

Scoliosis. It is our firm belief that permanent structural scoliosis will seldom if ever result if the empyema cavity is properly drained. Proper drainage will result in obliteration of the cavity with complete re-expansion of the lung. Postoperative *x*-rays taken during convalescence before the cavity had been obliterated often show some scoliosis. But at this time efforts should be directed towards favoring drainage and re-expansion of the lung, and the child should be encouraged to be as active as his condition warrants. Once the lung is well expanded and the cavity obliterated, the scoliosis will disappear. During convalescence methods directed toward the treatment of the apparent scoliosis *per se* are, in our opinion, contraindicated. Immobilization of the child on a Bradford frame, or some other form of apparatus, in order to treat the scoliosis will at this time hinder and may even defeat nature's successful efforts to overcome what is usually a functional and not a true structural scoliosis. Methods that aid nature in the obliteration of the cavity and re-expansion of the lung are the best methods of overcoming or preventing a scoliosis.

Anesthesia. Local novocain infiltration gives satisfactory anesthesia for aspiration and for most cases of intercostal closed drainage. Occasionally, a very brief general anesthesia is desirable for intercostal drainage. Nitrous oxide and oxygen if properly given is safe and satisfactory; cyclopropane is very useful but has the added risks inherent in its explosive nature. I still believe that ether and oxygen is at times superior to nitrous oxide and oxygen, and for a brief anasthesia we do not hesitate to use it in a chest case if we believe the anoxemia of even a short nitrous oxide administration is undesirable.

There is one general rule, however, that is most important. The patient whose condition does not warrant the use of a general anesthetic is the patient who at that time should not be submitted to those more radical forms of drainage for which general anesthesia is required and desirable.

Chronic Empyema. The best form of treatment of chronic empyema is its prevention by adequate treatment during the acute stage. The incidence of chronic empyema in cases under observation from the start at this hospital is as gratifyingly low as is the present low mortality rate. The treatment of chronic empyema is not within the scope of this paper. It, like chronic lung suppuration with which it is often associated, is a special problem.

Chemotherapy. As stated earlier, any definite conclusions on the usefulness of chemotherapy in the treatment of empyema cannot be made as yet. A very few patients with streptococcal empyema received sulfanilamide during 1938 which was the last year covered by the report from which statistical data have been quoted in this article. From January 1939 to June 1941 (two and one-half years) there have been forty-five cases of empyema treated at this hospital. If the same number enter in the next two and one-half year period, it will make a total of ninety cases for the first five-year period covering the use of chemotherapy. The two previous five-year periods gave an incidence of 150 and 137 cases, respectively. It is difficult to say whether this present number represents a true drop in the incidence of empyema because the incidence of empyema itself varies so much from year to year. We believe it is undoubtedly significant, however, that since 1939 of all the patients with acute pneumonia who entered the hospital and received chemotherapy, not one developed empyema. The patients with empyema treated here during the past two and one-half years entered the hospital with the empyema already developed. Some of these had had chemotherapy at home.

Our impression here is that sulfathiozole is superior to sulfanilamide even in streptococcal cases. Sulfathiozole seems superior to sulphapyridine in the pneumococcal cases. In recent months sulfadiazine seems superior to any of the sulfa compounds yet used and appears to have far less danger or cause less renal injury than sulfathiozole or sulphapyridine. Patients receiving chemotherapy demand careful observation with frequent estimation of the blood level of the drug, and particularly daily urinalysis to determine any evidence of kidney damage. Patients who have developed empyema under chemotherapy probably did not have the drug used early enough or in adequate dosage during the pneumonia, though this statement is not yet proved.

Treatment of an empyema that occurs following chemotherapy is worth discussion. It is probably of little use to continue chemotherapy once the empyema has been drained unless the pneumonic process itself is still active. A greater per cent of the patients with pneumococcal empyema who have had chemotherapy seem to require radical open drainage sooner in the course of the disease than did those in the past who did not have any chemotherapy. The exudate on the pleura is likely to be thicker and more tenacious and it is very difficult to clear up such a case by the closed methods. Although the number of cases is not large this has been a striking finding. It has been necessary to take greater pains actually to free with the finger the thick

tenacious exudate that lines the empyema cavity of such cases.

While it may be premature to say so, one gets the impression that if chemotherapy is properly used from the onset of the pneumonia, the incidence of a complicating empyema will be very slight. On the other hand if chemotherapy is not adequate in amount or is not started until late in the pneumonia, it is not yet possible to say that the use of this drug will lower the incidence of a complicating empyema. It is possible to say, although time may refute the statement, that cases receiving inadequate chemotherapy that do develop empyema, are more difficult to clear up. Our experience seems to indicate that open drainage will be used in a greater percentage of these cases than was the case before chemotherapy, but again we wish to emphasize that open drainage should not and need not be done as a primary procedure in patients still in the toxic stage of pneumonia.

CONCLUSIONS

The drop in the mortality statistics at this hospital before the event of chemotherapy justifies certain conclusions. Acute empyema should never be regarded as a surgical emergency. The condition in the pleural cavity should be treated as a complication of a general systemic infection. Children, especially infants, die with empyema and not because of it. The infecting organism should be determined in all cases by a diagnostic thoracentesis. The indication of each individual case must determine the proper type of drainage for that case. Local anasthesia for aspiration and intercostal closed drainage is indicated in almost all cases. Open drainage with or without rib resection is advocated in cases that do not respond after a period of time to closed methods of drainage. If the patient's condition contraindicates a general anesthetic, methods of drainage requiring general anesthesia are also contraindicated. Primary open drainage, with or without rib resection, should never be done during the synpneumonic stage or in children under two years of age. In properly selected cases primary rib resection is a safe and efficient method of treatment. If the empyema cavity is well drained and obliterated by the re-expansion of the lung, scoliosis need not be feared. It seems probable that the efficient use of chemotherapy will greatly lessen the incidence of a complicating empyema. While not yet proved, it would seem that if the empyema developed in patients receiving chemotherapy, its treatment required the more frequent and earlier use of open surgical drainage. It is likely that such cases did not have adequate dosage of the drug at the onset of the pneumonia.

ETIOLOGY OF CHRONIC EMPYEMA

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A PROPER understanding of the pathology and pathological physiology of acute empyema, and the application of these facts to the treatment of the disease would render chronic, nontuberculous empyema a clinical rarity. The findings of the Empyema Commission of the First World War are as valid today as they were in 1918; the facts have been emphasized in many publications, and yet we find daily instances in which these principles have been violated with disastrous results to the patient. It can be stated flatly that improper handling of acute empyemas is responsible for most cases of chronic empyema. There are some cases apparently doomed to chronicity from the onset, because of conditions which will be discussed later but these are a small percentage of the total. The incidence of chronic tuberculous empyema can be appreciably lowered, as the discussion will bring out. Since it can be seen that prophylaxis is of the utmost importance in the problem of chronic empyema, we believe it necessary to review the salient features of the pathology and treatment of the acute disease.

Empyema is the result of pleural reaction to infection, the extent and severity of the reaction varying with the infecting organism. In many cases of pneumococcic pneumonia it is probable that fluid is present in the pleural cavity before the crisis. Usually the amount of fluid is slight and the organization of fibrin produces agglutination of the pleural surfaces with subsidence of the pleural infection. If the process progresses and pus is formed, the abscess develops within the fibrinous deposit and may be large or small depending upon the amount of fluid and the presence or absence of limiting adhesions.

The route by which infection gains access to the pleural cavity has been variously discussed but the generally accepted opinion is that empyema develops as the result of the rupture of a small subpleural pulmonary abscess into the pleural cavity. Empyemas resulting from trauma to the chest and by extension from a subdiaphragmatic abscess are rare enough so that they may be disregarded as far as the purposes of this paper are concerned. Clinically, pneumococcic empyema develops after the pulmonary process has subsided. As a result the patient is suffering only from the effects of a localized, intrapleural abscess. In most cases fibrinous adhesions have formed between the previously involved lobe and the overlying parietal pleura. Along with this process fibrin has been deposited over the mediastinum, rendering it, as the fibrin organizes, a rigid septum between the two pleural cavities.

The pathological physiology of streptococcic empyema is quite different from that of pneumococcic. In the recognition of the difference and the effect this difference has upon treatment lies the secret of success in empyema. While in pneumococcic empyema the preceding pneumonia has usually subsided, in the streptococcic type the two conditions are concomitant. As a result the patient is much sicker and the mortality higher. It is believed that in streptococcic empyema the organisms spread through the interstitial pulmonary tissues to the pleura, producing a diffuse, abundant exudate. Because of the rapidity with which fluid develops and the comparatively low fibrin content of the fluid, limiting adhesions are unusual. Similarly, mediastinal stiffening does not occur.

As a result of the fundamental work of Graham and Bell certain facts of the utmost importance, particularly in regard to the effects of open pneumothorax, were brought to the attention of the profession.

They particularly emphasized that in the presence of a pneumothorax both lungs are affected nearly to the same extent. The loss of pulmonary function depends on the size of the pneumothorax opening. Of greatest importance is the relation of the size of the opening to the vital capacity. The greater the vital capacity the larger the opening tolerated. With a low vital capacity, approaching the point at which the maximum inspiratory effort only supplies tidal air requirements, a small pneumothorax opening may prove fatal. The above conclusions all suppose a thorax without adhesions or inflammatory change. In the presence of adhesions or a rigid mediastinum, as in chronic empyema, the normal pressure relationships no longer hold and the pressures in the pleural cavities may be changed independently. Therefore, large pneumothorax openings are well tolerated if adhesions or a rigid mediastinum are present.

The intelligent application of the fundamental facts of pathology and physiology, as noted above, are essential in the treatment of empyema so that mortality will be lowered and chronicity reduced to an unavoidable minimum. The methods commonly employed in the treatment of empyema, with many individual variations of technic, are aspiration, closed drainage and open drainage. The particular technic employed is unimportant, the attention to principles all important.

It has been said that in pneumococcic empyema the pulmonary lesion has usually subsided by the time the empyema becomes clinically evident. The pus is thick, adhesions are present and the mediastinum is rigid and indurated. In these cases open drainage is the treatment of choice. The establishment of an open pneumothorax offers little hazard to the patient since the vital capacity has not been lowered to dangerous levels and the rigid mediastinum prevents the altered pressure in the affected hemithorax from being transmitted to the uninvolved lung. We do not believe it essential that drainage be established at the most dependent portion of the empyema cavity. Attempts to reach the most dependent portion may result in damage to the diaphragm and the danger of peritonitis. Also, it has been found that rise of the diaphragm as the cavity becomes smaller may interfere with drainage. It is sufficient that drainage be established near or below the center of the cavity. Since an expanding lung increases in size concentrically, drainage in this area will be efficient.

As opposed to the treatment of pneumococcic empyema, open drainage should never be used in the course of a streptococcic empyema. An open pneumothorax must be avoided in these patients who are acutely ill, toxic and with greatly reduced vital capacities. The object of whatever method is chosen is to tide the patient over the period of acute pneumonia and to wait until intrapleural adhesions and a rigid mediastinum have formed, at which point open drainage may be established. This may be accomplished by either repeated aspirations or continuous closed drainage. The optimum time at which to institute open drainage will be determined by a fall in temperature, reduction in toxicity, relief of dyspnea and cyanosis and a change in the character of the pleural exudate from a thin, watery greenish material to a thick, creamy pus. If aspirations have been used, it is important that they be discontinued at this point, since lack of adequate drainage may cause great thickening of the visceral pleura, an unexpandable lung and a resultant chronic empyema.

The other commonly employed method of emergency treatment in streptococcic empyema is continuous closed drainage. This may be supplemented by some form of suction or tidal drainage depending on individual preferences. Both have had their enthusiastic advocates. It is doubtful if any form of closed drainage has much advantage over repeated aspirations followed by open drainage. It is very difficult to keep a closed system air-tight and the nursing care involved is a definite problem in the average hospital. It is our belief that the

chief value of closed drainage is as a substitute for aspirations in an extremely sick patient, provided that at the opportune moment open drainage be established.

It is apparent from the preceding remarks that chronic empyema will result most often from lack of attention to principles of treatment. Most important in the prevention of chronicity is adequate drainage, carried out at the proper time and continued until the cavity is obliterated. Drainage delayed until fibrin has organized and covered the visceral pleura with a thick, tough layer will prevent expansion of the lung and make for chronicity. Also, premature removal of drainage material before the cavity is obliterated will permit superficial healing of the drainage tract and maintenance of a residual pocket of empyema. It is in this type of case that we see the so-called recurrent empyemas which are, in reality, not recurrent since the initial empyema was never cured.

In attempting obliteration of an empyema cavity sterilization of the contents and liquefaction of the fibrinous exudate over the lung are essential. One of the most efficacious measures we possess to carry this out is Dakin's solution. Frequent irrigations with this material should be used in all cases in which there is no demonstrable bronchopleural fistula. Since many small bronchopleural fistulae close spontaneously as the empyema cavity closes, Dakin's solution may be cautiously re-employed at intervals if a fistula has necessitated its temporary discontinuance. All empyema cavities should be carefully measured and frequently examined roentgenologically, employing a contrast medium such as lipiodol, if indicated. Only when the capacity of a cavity is a half ounce or less should drainage be removed.

In addition to the factors of improper, delayed or insufficient drainage as causes of chronicity, the causes usually given are tuberculosis, persistent bronchopleural fistula, underlying lung abscess, intrapleural foreign bodies, osteomyelitis of ribs and underlying carcinoma of the lung. Butler has reported several cases in which another predisposing cause of chronicity may play a part, namely, postpneumonic atelectasis. Hochberg has recently reported two cases in which this mechanism seemed to play an important rôle.

Tuberculous empyema is a chronic disease; its most effective treatment is prevention. We believe that the incidence of tuberculous empyema can be materially reduced. It is known that the percentage of effectiveness of pneumothorax decreases in direct proportion to the time taken for the pneumothorax to become effective. It is also a fact that the majority of cases of tuberculous empyema follow as a complication of pneumothorax. More empyemas develop in ineffective pneumothoraces than in effective ones. Therefore, if ineffective pneumothoraces were either abandoned promptly or abandoned as soon as section of adhesions by closed, internal pneumonolysis was found impossible, the number of cases of tuberculous empyema would rapidly drop.

Persistent bronchopleural fistula as a cause of chronicity can be avoided in some cases by proper drainage. Delay in instituting drainage is responsible for a few persistent fistulae. However, many of these cases fall into the category of inevitable chronicity. Prompt recognition and treatment of pulmonary abscesses will eliminate many cases of chronic empyema resulting from neglect of this condition. It is our belief that in large lung abscesses, involving a large portion of a lobe, lobectomy rather than drainage will be the treatment of the future.

Foreign bodies retained in the pleura are usually either pieces of rubber tubing or gauze. That these are preventable requires no argument.

Osteomyelitis of ribs is mentioned in every series of cases of chronic empyema. While it occasionally occurs as a cause of chronicity, we believe that more often it results from chronicity and that there are other factors accountable which might be found if carefully searched for, not only

in the chest at the time of operation but also in the history of the original treatment.

The prevention of chronic empyema due to carcinoma of the lung lies of course in early diagnosis and treatment of this increasingly prevalent tumor.

The interesting conception of Butler, previously mentioned deserves some attention. In the four cases reported by this observer and the two mentioned by Hochberg, delayed closure of an empyema pocket and an unusually stormy course seemed directly related to bronchial obstruction and atelectasis. If this condition is suspected roentgenography, pneumonography and bronchoscopy should be done. Dramatic improvement and avoidance of chronicity may be expected if the bronchial obstruction is relieved. Butler believes that even partial relief of the obstruction may allow complete aeration. This is based on the work of Van Allen and Jung who demonstrated that collateral respiration may take place in a lobe in the presence of obstruction of some of the smaller bronchi due to the openings in the interlobular septa and alveoli.

Since we have been interested in the prevention of chronic empyema we have reviewed the last fifty cases of this condition admitted to the Jefferson Hospital. Of these, eight were tuberculous and may be eliminated from the analysis. In many of the cases, most of whom had had their original treatments elsewhere, the histories of the initial illness were not available and conclusions could be drawn only by inference. In ten of the cases the condition could be specifically traced to improper, neglected or insufficient drainage. In fifteen, although the data were incomplete, the histories were highly suggestive of the same factors. Excluding the tuberculous cases it will be seen that 60 per cent of the chronic, nontuberculous empyemas were attributable to the neglect of the fundamental principles of treatment of acute empyema. In twelve of the cases bronchial fistulae were given as the cause of chronicity. However, since it is likely that most cases of empyema have bronchial fistulae some time during the course of the disease and that most of these close spontaneously with proper treatment, it seems reasonable to predicate that in at least a few of this group the persistent fistulae were an expression of improper treatment rather than a primary cause of chronicity. Two cases were due to foreign bodies and three were attributed to osteomyelitis of ribs. We have previously stated our opinions upon these subjects.

SUMMARY

The fundamental features of the pathology and physiology of acute empyema have been reviewed.

The application of these principles in the treatment of acute empyema have been noted.

The factors leading to the development of chronic empyema have been explained.

Fifty cases of chronic empyema have been analyzed, particularly as to etiology.

CONCLUSIONS

1. Application of the fundamental principles of pathological physiology is essential in the treatment of acute empyema if chronic empyema is to be avoided.

2. Neglect of these principles is responsible for the majority of cases of chronic empyema.

3. This was borne out by an analysis of fifty cases of chronic empyema from the Jefferson Hospital.

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ENCAPSULATED EMPYEMA

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ANY collection of pus within the pleural space, which does not occupy the whole space, can be regarded as a localized empyema in the broadest meaning of the term. For clinical purposes, however, the terms, "localized" or "encapsulated" or "sacculated" are generally applied to smaller collections of pus that are completely sealed off from adjacent uninvolved pleura. It appears to us that special consideration of the localized or encapsulated empyemas is warranted because (1) the pathogenesis, evolution and clinical features often are different from those of total or diffuse empyema; (2) the roentgenographic features are different and problems in roentgenographic diagnosis arise which do not exist ordinarily in total empyema; (3) there are special problems connected with exploratory thoracocentesis; and (4) the surgical management often requires a distinctive approach. Before proceeding with the discussion of these four aspects of the subject, it should be stated that very large, so-called subtotal empyemas usually resemble total empyemas so closely that they will not be considered. However, it may not be amiss to point out that the uninvolved pleural space may become infected at operation even in such cases, if certain principles to be set forth concerning diagnosis and surgical management are not observed.

PATHOGENESIS, EVOLUTION AND CLINICAL FEATURES

The question of the pathogenesis, evolution and clinical features of localized empyema should be considered first because of distinctive features which we believe to exist in comparison with those of total or diffuse empyema. At the present time it is generally recognized that the cause of suppurative pleuritis in a substantial proportion of cases is an underlying suppurative pulmonary focus. The latter is unequivocally proved to be present in cases of pyopneumothorax. In cases of empyema the existence of a causative pulmonary abscess may be established at operation, in the postoperative course, at secondary operations, etc. Thus, the absence of air (fluid level) in a case of localized empyema does not negate a causative suppurative pulmonary focus. This point warrants emphasis because a study of our cases of localized empyema discloses the fact that the vast majority are due to suppurative pulmonary foci, usually perforated pulmonary abscesses. This held whether or not roentgenograms revealed the existence of a pyopneumothorax and regardless of the presence or absence of a perforated pulmonary focus at the time of operation. By way of contrast it can be said that the majority of cases of total and diffuse empyemas are not due to demonstrable suppurative pulmonary foci. Another feature which distinguishes the two lesions is the extent of the pulmonary abscess. When present in cases of diffuse empyema, it is usually small and insignificant and apparently an incidental part of a more or less widespread pneumonic lesion. On the other hand, the pulmonary focus is a substantial localized abscess in not a small proportion of cases of encapsulated empyema. Indeed, the causative pulmonary abscess may be larger and more significant clinically than the pleural empyema. Finally, there are instances of encapsulated empyema, such as occasional cases of ruptured interlobar abscess (so-called interlobar empyema), in which involvement of the pleura is so limited that its existence can only be recognized if specially sought for.

From the foregoing remarks on pathogenesis as well as from a consideration of

the limited extent of pleural involvement in many cases, one can anticipate that the evolution of localized empyema would often be much more insidious and much less obvious than that of diffuse and total empyema. Indeed, the existence of the lesion is scarcely to be suspected in a substantial proportion of cases until revealed by roentgenograms. The symptomatology of encapsulated empyema is in keeping. In some instances the symptoms are essentially those of the underlying abscess whether putrid (anaerobic) or aerobic. Invasion of the pleura is initiated in such cases by a sudden access of localized thoracic pain and may be characterized (by no means invariably, however), by reduction in cough and purulent expectoration. When symptoms of pulmonary abscess do not exist, the course usually is that of an acute pneumonic lesion in the initial phase, followed by low grade fever. Only rarely are there seen the evidence of respiratory distress, cardiac embarrassment or toxemia as often noted in diffuse empyema. This statement applies to children as well as to adults. Indeed, the patient may be up and about for a long time (weeks or even months) before the encapsulated empyema is discovered. A number of our cases first came under observation so long after the acute pulmonary episode that pulmonary neoplasm and not localized empyema was the first diagnosis made on the basis of a well delimited shadow in the roentgenogram.

It is evident that both the physical features and the symptomatology of encapsulated empyemas usually are less simple and clear than those of diffuse empyemas. Indeed, an encapsulated empyema can be defined as a lesion characterized by atypical manifestations. Several features of encapsulated empyema warrant special mention. First, the empyemas which may have little or no parietal contact should be mentioned particularly because of difficulties encountered in diagnosis and of danger of infecting the free pleural cavity by exploratory aspiration or at operation. There are three groups of such cases: The interlobar (which we have shown to be perforated pulmonary abscesses[1,2]) the infrapulmonary and the paramediastinal. At times empyemas in other situations also may present limited contact with the chest wall, apparently because firm parietal adhesions over a small area lead instead to indentation of the lung by the collection of pus. Another feature of localized empyema worthy of special mention is multilocularity. Two or more intercommunicating loculations are common. In some instances loculations appear far removed from one another, but intercommunication by more or less narrow pathways is the rule. Localized pleural invasion as the result of the perforation of a pulmonary abscess of substantial proportions requires discussion because of the problems in diagnosis and surgical treatment which are raised. The symptoms and diagnosis of pulmonary abscess cannot here be described. For our present purposes it will suffice to say that a pulmonary abscess is characterized by the expectoration of pus. Apparently there is a general impression that the perforation of a pulmonary abscess into the pleura is signalized by the cessation of purulent expectoration. Associated with this impression is a belief that the perforation is likely to result in cure of the abscess. Evidence has been advanced elsewhere to show that neither view is tenable.[3,4] Spontaneous cure can follow perforation and expectoration of pus may cease after perforation, but neither phenomenon can be anticipated with any assurance in cases of perforations of substantial abscesses into localized spaces. Thus, the absence of perforation cannot be postulated if expectoration of pus continues and the assumption of cure of the abscess is not warranted because perforation has occurred. The differentiation between the presence and absence of perforation, obviously important for clinical reasons, must be made chiefly by radiological examination as will be shown.

Another feature of encapsulated empyema is the frequent occurrence of adjacent bland pleural effusions, so-called sympathetic effusions. When such effusions encountered by exploratory aspiration present evidences of infection, they may be mistaken for the underlying localized collection of pus. The recognition of their existence in cases of encapsulated empyema is therefore of importance. Finally, reference should be made to a more or less chronic, localized empyema which masquerades as "thickened pleura." This lesion, characterized by fever, dullness to circumscribed flatness on percussion, usually distant breath sounds and a roentgen film of localized density is now generally recognized as an encapsulated empyema. In fact, the pleurae usually are greatly thickened and the collection of pus may be small. Not so generally known is the fact that this type of lesion may follow a variety of intrathoracic operations. Indeed, it may be encountered after operations for nonsuppurative lesions, presumably as the result of unrecognized infection or infection of retained blood. Of particular importance is the variety which may be termed a residue after a more widespread pleural infection has been drained. In such cases the patients may appear to be essentially well for months or even years, complaining only occasionally of episodes of thoracic discomfort or pain and slight transient fever. The roentgen films, however, will always disclose the appearance of "thickened pleura" even during the periods of complete well being.

ROENTGENOGRAPHIC FEATURES AND DIAGNOSIS

A quantity of pus in the pleural cavity casts a dense homogeneous *x*-ray shadow. While the boundaries of total or subtotal empyema are obscured on the films, at least one boundary will be clearly defined in the case of encapsulated empyema. If a pneumonic shadow exists, the collection of pus usually casts a homogeneous shadow which is denser and which usually has at least one well defined margin. The shadow of an encapsulated empyema is usually semi-ovoid in shape with the base against the chest wall. The inner margin may be straight, convex mesially or scalloped if the collection is in the axilla. If situated at the anterior or posterior chest wall, only lateral or oblique views will demonstrate limits of loculation. A homogeneous density over pneumonic lung which lacks more or less clearly defined margins distinct from those of pneumonitis is probably due to plastic pleural exudate. Only re-examination can determine if pleural pus with loculation will supervene. The variations of these fundamental roentgen features will be discussed under the separate caption devoted to special varieties.

Throughout the discussion the terms encapsulated or circumscribed empyema and encapsulated or circumscribed pyopneumonothorax will be employed interchangeably. The reason is to be found in the fact that from the viewpoint of roentgenology a fluid level is in the nature of an accident. Its presence is indubitable evidence of a perforated bronchogenic pulmonary abscess. The absence of a fluid level does not exclude the existence of a perforated abscess. The absence of air in the empyema may mean nothing more than the absence of a free bronchial communication at the time of perforation of the abscess. Attention has already been directed to the frequency of perforated pulmonary abscess as the cause of encapsulated empyema. We may add here that, in not a few instances, the existence of the perforated pulmonary focus is to be noted for the first time at operation (or after operation by the discovery of a bronchial fistula), because preoperative films failed to reveal fluid levels or air pockets within the shadows of encapsulated empyemas.

As already indicated the diagnosis of an encapsulated empyema must be based chiefly on the roentgenological examination. Of physical signs the most important is localized dullness or flatness on precussion, an abnormality which is better

identified by the sense of touch than by sound. The roentgen film depicts accurately the nature of the process in the vast majority of cases, and errors in diagnosis are to be ascribed to errors in interpretation and not to shortcomings of the method. Only rarely will obscuring or overlying shadows render impossible a satisfactory study of the pleural encapsulation. In our experience almost all errors in roentgen diagnosis could be recognized as such after the existence of encapsulated empyema was disclosed by operation if re-inspection and restudy of the films were made. The reverse was also true, namely, the absence of encapsulated empyema, erroneously diagnosed on roentgen examination, can also be established by study of the films.

The outstanding exception to the foregoing statements is to be found in the roentgen diagnosis of encapsulated empyemas (or pyopneumothorax) due to perforation of pulmonary abscesses of substantial proportions. In not a few instances we have operated on the roentgen diagnosis of perforated pulmonary abscess to find the abscess of the lung unperforated at the time of operation. It is our impression that the roentgen diagnosis cannot be made at times. Thus, when a pulmonary abscess faces an interlobar fissure with resultant pronounced interlobar pleuritis, the differentiation between rupture and nonrupture into the fissure appears impossible. However, the diagnosis of perforation of an abscess can be made in most instances on the basis of two features: (1) The existence of a considerable area of homogeneous density with sharply defined convex margin, and (2) the existence of two (or more) adjacent areas of rarefaction as well as of two (or more) fluid levels. Should an earlier film have revealed an uncomplicated pulmonary abscess, later films would, of course, establish the diagnosis if the pleural complication were depicted. (Fig. 1.)

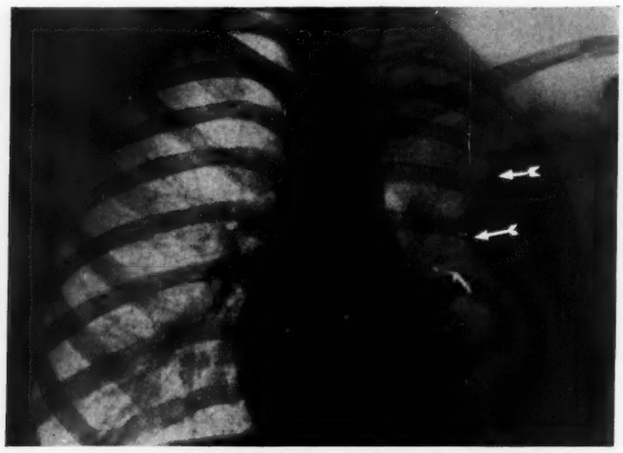

Fig. 1. Loculated lateral empyema. The collection, situated in the axilla, extends over the apex of the lung (confirmed at operation). Note the sharp anterior and posterior margins (arrows). Note also the scoliosis, not uncommon in empyema in children.

Turning now to the special roentgen diagnosis of encapsulated empyemas, it can be said that purulent collections may occur anywhere in the pleural cavities. They are found most often in the general pleural space, that is, outside interlobar fissures. Because their locations present peculiar problems in diagnosis and surgical approach, five groups will be discussed separately. As a statement applying to all groups it should be said that the diagnosis of the existence as well as the extent of encapsulated empyemas in any of these groups may depend on oblique, lateral or other views in addition to the conventional postero-anterior films. This is particularly

true of small encapsulations in atypical places such as the retrocardiac, supradiaphragmatic or mesial regions.

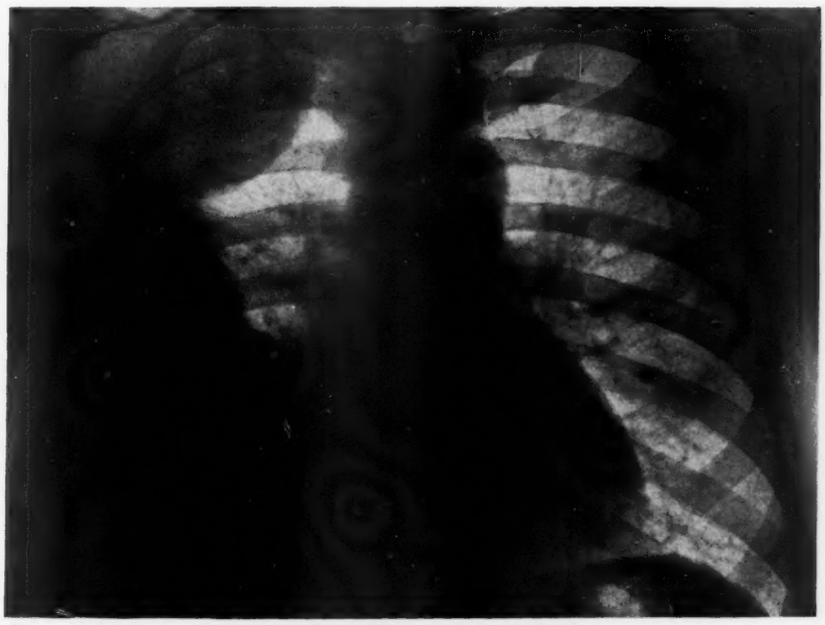

Fig. 2. Bilocular empyema. One collection is subapical and the other, lateral. Note the characteristic sharp delimiting margins.

A. Encapsulations over the convexity, which are the most common, are located on the anterior, posterior or lateral aspects of the convex curve of the chest wall.

B. Mediastinal. These occur on the mesial or mediastinal aspect of the general pleura.

C. Supra- or periapical surmount or surround the apex of the lung.

D. Infrapulmonary. These occur between the undersurface of the lung and the uppersurface of the dome of the diaphragm.

E. Interlobar are loculated between opposing surfaces of the interlobar pleura. (Fig. 2.)

A. Collections over the Convexity. These empyemas vary in size from large collections to small pockets containing but a few cc. of pus. The smaller collections usually present no great difficulty in diagnosis. Occurring in conjunction with the shadow of a pneumonic lesion and revealing well defined margins, the diagnosis is usually evident. They may, however, be obscured by a large pneumonic lesion with surrounding abundant plastic exudate or by a sympathetic effusion. In the case of very large encapsulations the roentgen diagnosis may be difficult or even impossible if pleural delimitation of a homogeneous shadow is not visible.

Multiple views are imperative in order to disclose loculation if a homogeneous shadow is present with consolidation of the lung.

Bizarre appearances are often seen in cases of multiloculated empyema. If surgical drainage has already been instituted, repeated radiographic examinations may be necessary to determine if loculations intercommunicate or to disclose other collections previously unrecognized. When pyopneumothorax occurs following the rupture of a lung abscess, even fluoroscopy and films made in many projections, with the patient in both the erect and lateral recumbent positions, may not suffice to distinguish between an intrapulmonary or an intrapleural lesion. Suggestions for differential diagnosis have already been made. (Fig. 3.)

B. Paramediastinal Empyema. These collections are situated on the mediastinal aspect of the pleura, immediately adjacent to the anterior, middle or posterior medias-

tinum. Because of the superimposition of shadows on the heart, great vessels and other mediastinal structures, unusual pictures are produced which are often difficult of correct interpretation. However, the diagnosis by roentgenology can be made in the majority of instances if the essential features of localized empyema, to which attention has been called, are borne in mind.

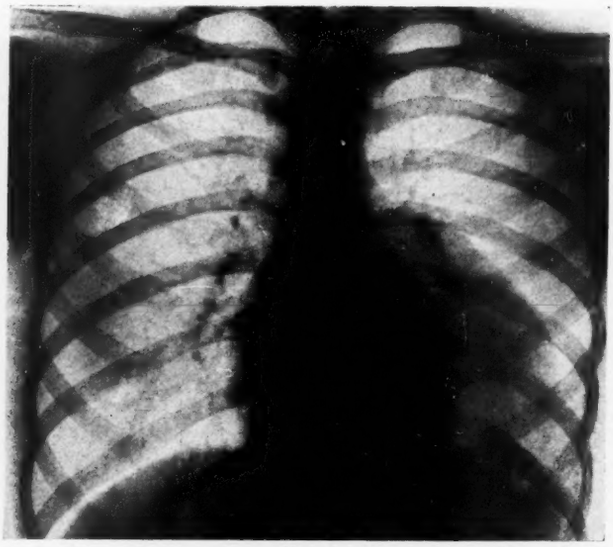

Fig. 3. Large lower paramediastinal empyema. The technic used demonstrates the separate overlapping shadows. The appearance might otherwise simulate an enlarged heart.

Some of the problems in diagnosis may be cited. When the empyema occurs in the lower mediastinum, the appearance may simulate cardiac enlargement or localized pericardial effusion. This is especially true if the exposure technic results in films with marked contrast, so that the overlapping cardiac and pleural shadows cannot be separated.

When the empyema occurs in the upper mediastinum it may simulate, on the postero-anterior projection, an aortic aneurysm or mediastinal tumor. If the loculation extends laterally enough, its anterior and posterior margins may be sharply defined on the lateral projections. In the case of a shallow empyema the longest diameter often is in the sagittal plane; its anterior and posterior adhesions will not be clearly visualized in contrast with mediastinal shadows.

The differentiation between a large paramediastinal empyema and a mediastinal abscess may be impossible roentgenologically. A lead may be found in a sympathetic pleural effusion or in a second pleural loculation. Occasionally, a pulmonary abscess facing the mediastinum may rupture into that potential space producing an encapsulation which is impossible of roentgen differentiation from paramediastinal empyema. (Fig. 4.)

C. Supra- or Peri-apical Empyema. This variety of empyema is rare. In the cases we have seen it has been the result of a rupture of a pulmonary suppurative focus or a loculation separated by adhesions from a generalized empyema which had extended over the apex. In two instances a supra-apical empyema acted as a cowl over the top of the lung, requiring both anterior and posterior upper thoracic drainage. The *x*-ray film shows a homogeneous shadow extending downward for a variable distance from the dome of the hemithorax. The inferior margin of the shadow is usually sharp in outline. No air-bearing tissue is seen above this shadow. While the appearance may be considered to be characteristic, we have seen identical roentgen evi-

dence in cases of apical pneumonia with plastic exudate, atelectatic upper lobe in a child, and in pulmonary abscess.

Malignant neoplasms occupying the apical and paravertebral portions of the upper lobe may present features closely resembling those of apical or peri-apical empyema. Destruction of overlying ribs, usually visible in roentgen films, often establishes the diagnosis. The differentiation from empyema may be particularly difficult if secondary pleural infection or carcinomatous pleural invasion supervenes.

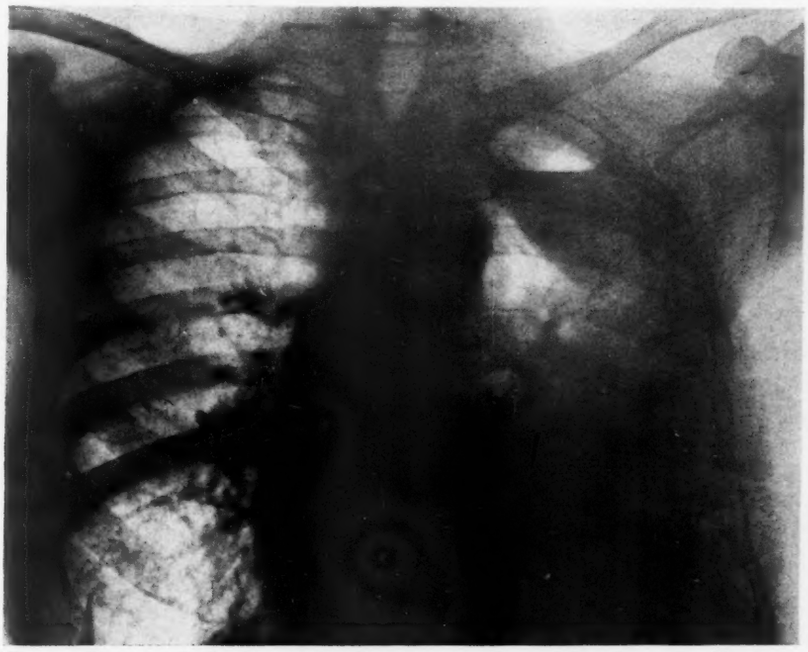

Fig. 4. Supra-apical pyopneumothorax, occurring with a lateral collection. Note the absence of lung markings at the apex.

D. Infrapulmonary Empyema. This term is applied to an encapsulated collection of pus situated between the under surface of the lung and upper surface of the dome of the diaphragm.[5] Infrapulmonary empyemas vary in extent from lesions which are small and are entirely separated from the chest wall by adherent lung, to the larger collections which encroach on the pleura of the thoracic convexity.

When the lesion is small, it is apt to be overshadowed by adjacent pulmonary infiltration. Larger localized infrapulmonary encapsulations usually elevate and arch the diaphragmatic surface of the lung. As a result, a shadow is present at the base of the lung merging with the density of the liver (on the right side). The *x*-ray appearance may be indistinguishable from that of subdiaphragmatic abscess.

As already noted, an infrapulmonary empyema of large size usually extends into the general convex portion of the pleural space, generally in its posterior aspect. If the parietal extension is small, it may be overshadowed by the original collection; while if large, it may obscure the infrapulmonary lesion. Lateral films may aid greatly in resolving the shadows. (Fig. 5.)

When an infrapulmonary pyopneumothorax is present, the infiltrated compressed lower lobe is usually elevated by a collection of air surmounting the fluid level. This appearance is difficult to differentiate from a gas-containing subphrenic abscess. The induction of a small pneumoperitoneum may delineate the dome of the diaphragm and thus disclose the situation of the lesion. The same procedure may aid in the differentiation of infrapulmonary lesions which do not contain air.

A useful sign of an infrapulmonary empyema, not present in all cases, is the displacement of the heart away from the side of the lesion. This sign is particularly

valuable when the empyema is small and may be mistaken for pneumonia.

E. Interlobar Perforated Pulmonary Abscess ("*Interlobar Empyema*"). As we have shown elsewhere[1,2] all cases of true interlobar collections of pus have proved, in our experience, to be cases of perforated pulmonary abscess. The latter term can, therefore, be applied properly to the group of cases in which pus is encapsulated in interlobar fissures. About half of the cases in our series presented roentgenological evidence to indicate the presence of interlobar disease. Since it is important to recognize the existence of an interlobar abscess for purposes of accurate surgical approach and management, a statement of the positive roentgen features is warranted.

An oval or elliptical shadow limited by the interlobar fissures, or overlying the region of the fissures, or the presence of a pyopneumothorax limited by the interlobar pleurae is strongly suggestive evidence. However, only the latter film can be regarded as pathognomonic for we have seen apparent intrafissural lesions prove at operation to be due to pulmonary infiltration.

A fluid level abutting on, or traversing, a fissure should also suggest the possibility of an interlobar collection. Bowing of a fissure, due to pressure by a large interlobar collection of pus was noted in one of our cases. In another case, in films taken at intervals, there was seen the progression of disease along an interlobar fissure. This was found to be due to an interlobar abscess which had gradually enlarged by dissecting along the interlobar pleural space.

A B

FIG. 5. A, anteroposterior view of infrapulmonary empyema. The appearance simulates an elevated left dome of the diaphragm, possibly as a result of subphrenic abscess. B, lateral view. The lateral projection illustrates the large posterior parietal presentation.

In about half the cases we reported there was no distinctive roentgenological evidence to suggest the diagnosis. In some the lesion was obscured by large pleural effusions; in others, the pulmonary lesion dominated the picture. There was one instance in which a sharply circumscribed shadow seen in the roentgenogram was thought to be due to a pulmonary neoplasm.

THORACOCENTESIS FOR DIAGNOSIS

The foregoing discussion on various aspects of the subject of encapsulated empyema indicates clearly that the diagnosis is difficult in not a small proportion of cases. As compared with diffuse empyema in which thoracocentesis often is performed only for confirmation of the diagnosis, exploratory puncture is not infrequently required in an effort to establish the diagnosis of encapsulated empyema. Indeed, there are instances in which very carefully performed exploratory aspirations fail to reveal pus, cases in which exploratory thoracotomy is indicated rather than to subject the patient to indiscriminate (and possibly dangerous) multiple aspira-

tions. Since the diagnostic thoracocentesis evidently occupies a vital place in the management of encapsulated empyema, it should be performed under the most favorable auspices. The latter implies first, readiness to proceed with operation if pus is encountered, the needle being left in place if the empyema is small or difficult to find by aspiration, and secondly, careful roentgenological study. Encapsulated empyema rarely is a lesion urgent for operation and ample time, therefore, is available for complete roentgen films taken in the various positions required by the individual case. Only then will the correct intercostal space for exploratory puncture be disclosed, namely, the site at which there is contact between the empyema and the thoracic parietes. Since absence of such contact is decidedly unusual, as has already been stated and since adequate roentgen studies will reveal the precise site of contact, pus should not only be encountered promptly in most instances but also superficially. Deep aspiration is warranted only in the unusual instances already referred to (interlobar, infrapulmonary, paramediastinal, high axillary empyemas) in which the main collection of pus is not in contact with the thoracic convexity. It is also evident that deep aspiration may reach an underlying pulmonary abscess and not the empyema. Finally, reference should be made to the fact that the diagnosis of empyema is not necessarily established when only small amounts of pus are obtained by aspiration. Errors in diagnosis have been made since such small quantities may be obtained directly from the infected lung. The danger of infection of a previously uninfected pleural space under such circumstances is obvious. That errors have been made has been proved at operation, in cases in which the pleural space has been found to be free from any suppurative lesion.

SURGICAL TREATMENT

The prerequisite for correct operative treatment of sacculated empyema is precise roentgen localization of the lesion with special reference to its contact with the thoracic parietes. The essential step in the operative procedure is adequate exposure of the parietal pleura in order to visualize the presence (or absence) of pleuritis at the site of proposed entry into the collection of pus. Obviously, we regard entry of the free pleura as the outstanding danger of operation for encapsulated empyema. It can be said that this danger scarcely exists when encapsulations are of large size. Yet we know of instances in which the general pleural space has become infected as the result of being entered in such cases. In any event, in the case of moderate sized, or more particularly of relatively small encapsulations, the danger is real unless preoperative localization is accurate and the parietal pleura is visualized at operation. As has been pointed out, the free pleura must of necessity be traversed in specified, relatively rare instances in order to gain access to collections of pus. With the knowledge that slight, if any, contact is present in such cases the free pleural cavity can be entered, packed off and traversed in a one- or two-stage operation without becoming infected.

In our opinion, closed drainage has little or no place in the operative treatment of encapsulated empyema, even in children. There are three reasons for our view: First, the well known indications for closed drainage rarely exist in cases of localized empyema. It can be added that closed drainage rarely would be curative for encapsulated empyema, and would therefore be only a preliminary procedure. Secondly, closed drainage is too blind a procedure for localized empyema, the danger of infection of the free pleural cavity existing unless the empyema is of large size. Thirdly, closed drainage not only is unnecessary but is almost certain to be ineffective for adequate drainage, particularly in the presence of loculations of pus or of an underlying perforated pulmonary abscess of substantial size.

Local anesthesia is the anesthesia of choice for operations for encapsulated empyema; only rarely should supplement-

ary general anesthesia be necessary. There are two special advantages in local anesthesia: first, the danger of spillover infection to other pulmonary segments is minimal in the event of underlying pulmonary abscess communicating with the bronchial tree; secondly, the patient can cough or strain on request during operation and as a result reveal a pocket or pockets of pus which otherwise might be overlooked. If inhalation anesthesia must be used, positive pressure can be employed to disclose such pockets.

The liberal excision of a portion of one rib usually suffices for adequate exposure of the pleural abscess provided the correct rib has been chosen. After the collection of pus has been entered the parietal pleura should be split open to unroof the empyema completely, if possible. In so doing every precaution should be exercised to avoid entry of the free pleural space, and reliance should be placed on visualization rather than on sound because the hiss or blow of an open pleura resembles closely that of an open bronchus.

After the main collection of pus has been laid open satisfactorily and evacuated by suction an examination is made for communicating locules. The roentgen film may have already demonstrated their existence and situation and the direction in which they are to be sought has, therefore, been indicated. However, additional pockets may be found in cases in which there is no preoperative evidence to indicate their existence. It is important to note that communications between locules and the main cavity may be via narrow and tortuous channels sometimes situated in relatively inaccessible recesses of the parent abscess. The use of a sterile light for the examination for recesses and communications is invaluable. Coughing or straining by the patient may reveal a pocket which otherwise might be overlooked. Ramifications from the primary cavity can usually be laid open from within but may require enlargement of the wound with additional rib removal. At times counterincisions are necessary, particularly when secondary pockets are in contact with the thoracic parietes at sites remote from the original incision. A single cavity should be the objective of operation and can be achieved in the preponderance of cases. Under such circumstances the cavity and any recesses which exist can be packed with gauze at the end of operation. Tube drainage in addition to gauze packing of the main cavity should be reserved for those exceptional instances in which wide opening of the main or secondary collections of pus has not been carried out.

The relationship between pulmonary abscess and encapsulated empyema was taken up in the first section. The operative treatment of the pulmonary abscess should now be discussed. It should be stated at the outset that a unanimity of views does not exist at the present time. There is the widely held view, to which reference has been made, that pleural perforation of a pulmonary abscess may be or is tantamount to cure of the abscess, and the obvious corollary that operative treatment of the abscess is not indicated. We believe that the latter applies to many cases but that rigid adherence to such a plan is dangerous. Thus, there are not a few cases in which the pulmonary abscess is of substantial size and the perforation insignificant. In such cases the pulmonary symptoms may be prominent or may even dominate the clinical picture. In our opinion, such pulmonary lesions should be cared for at the time of operation for empyema. The justification for this view is to be found not only in the desirability of eliminating the symptoms of pulmonary abscess but also in the extreme simplicity of the operative procedure. The latter consists in visualizing the opening in the pulmonary abscess, splitting it crucially if found to be too small and inadequate for drainage of the pulmonary cavity, the insertion of narrow-bladed retractors and inspection of the interior of the cavity for recesses, and the gauze packing of the pulmonary cavity and its recesses after it has thus been adequately unroofed. Only rarely will a patient's condition be too poor

to withstand this simple and brief additional operative procedure.

Special reference should be made to the subject of putrid empyema in connection with the question of management of the underlying abscess. It has been shown elsewhere[3] that an abscess is to be demonstrated as the almost invariable cause of an encapsulated collection of foul pus in the pleura. When profuse expectoration of foul pus is a symptom, a pulmonary abscess of substantial proportion can be anticipated. The pulmonary lesion may be seen in films which antedated, as well as in those taken at the time of pleural invasion. In our opinion, adequate unroofing of the perforated pulmonary abscess is imperative under such circumstances. Putrid pulmonary abscess is a far more dangerous lesion than aerobic or nonputrid pulmonary abscess and temporization, which may be warranted in the one is unjustified in the other. In either instance the possibility of spontaneous recovery exists. In the case of a putrid lesion, however, the possibility should not be banked on. The argument in favor of deferring the unroofing of a perforated abscess to a later date in order to ascertain if the procedure proves to be indicated can be refuted on three counts: (1) During the interval there may be a spread of the anaerobic (putrid) infection beyond the limits of the pulmonary abscess not only locally, but also in remote segments. (2) Spread of infection may occur insidiously with few, if any, symptoms of pulmonary abscess and without characteristic roentgen features. (3) Unroofing at the time of the primary operation is simple and safe, whereas the abscess may be difficult to find and not simple and safe to drain at a deferred operation.

The immediate results of primary unroofing of perforated abscesses at the time of operation for putrid empyema are striking in the prompt recession of the symptoms of pulmonary abscess. Furthermore, symptoms do not return if the pulmonary cavity is not permitted to close prematurely. Special post-operative attention to the pulmonary cavity is required after an abscess has been unroofed. The cavity should be kept open by packing and the bronchial fistula must be maintained until all evidence of anaerobic infection has disappeared. Infection within the pulmonary cavity may persist after the empyema space has become quite clean. Therefore, the opening in the lung may prove to be the last part of the wound that is kept open. The foregoing remarks were made specifically in reference to perforated putrid abscess but apply also in a general way to the postoperative management of perforated aerobic abscess.

Although the operative treatment of the pulmonary lesion comprises the most distinctive feature of the surgery of encapsulated empyema, the basic principle of treatment is complete drainage of the suppurative focus without infection of the free pleural space. There is a rather general belief that the results of operation for empyema are related to the underlying cause rather than to the operative technic. This belief is justified in some cases but cannot be enunciated as a dictum. Not only morbidity but also mortality can be directly ascribable to a badly conceived or incorrectly executed operation for encapsulated empyema. On the other hand, uniformly good results can be anticipated, unless the underlying cause is irremediable surgically (pulmonary cancer, actinomycosis, etc.), if operations are based on accurate preoperative studies and are carefully executed in accordance with a well conceived plan.

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BRONCHIECTASIS*

MORBIDITY AND MORTALITY OF MEDICALLY TREATED PATIENTS

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THE pathology of bronchiectasis was described in considerable detail by Laennec more than a century ago. However, a fairly precise knowledge of the etiology, pathogenesis, bacteriology, and the morbidity and mortality of the disease, has been accumulating only during the past seventeen or eighteen years, or since Sicard and Forestier first used iodized oil in bronchography. Despite the rapid advances in our knowledge and concepts of bronchiectasis during these recent years, considerable differences of opinion still exist regarding many aspects of the disease. Mueller, in 1928, and Ballon, Singer and Graham, in 1931, and more recently, Miller and others, emphasized the congenital nature of bronchiectasis; however, Perry and King, Warner, McNeil, Findlay and Sauerbruch, to mention but a few, have expressed considerable doubt regarding the congenital nature of the condition. In a large series of cases observed at Bellevue and Lenox Hill Hospitals during the past ten years, no proved case of congenital bronchiectasis has been seen by the writer. Occasional writers still emphasize the rôle of spirochetes, although the majority of workers in this field minimize their importance in bronchiectasis.

Of greater practical importance is the differences of opinion regarding the treatment of bronchiectasis. In many clinics where large series of cases are being collected, followed and studied, a more unified and clear conception of the natural course of the disease and its treatment is emerging. Nevertheless wide differences of opinion still exist as evidenced by reports in the literature by members of the various specialties, extolling different methods of treatment. These differences in point of view between certain internists, roentgenologists, bronchoscopists and surgeons, apparently spring from differences of understanding of the fundamentals of the disease—its etiology and pathology, pathogenesis and pathological physiology, bacteriology, the varied clinical patterns, its usual course characterized by natural remission of symptoms for long periods of time, frequently to be followed by exacerbation and progression of the disease, often eventually resulting in hopeless chronic invalidism if the proper treatment is not rendered at the opportune time. Bronchiectasis is often similar to pulmonary tuberculosis in that it tends to relapse and progress; and also most cases of bronchiectasis, like most cases of tuberculosis, are potentially curable at some phase of the disease. Often, circumstance, patient and doctor contrive to overlook this opportune moment and fail to choose the best method of treatment.

The internist who hopefully administers arsenicals because of the erroneous conception that spirochetes constitute the principal bacteriological agent in the usual case of bronchiectasis, apparently lacks a comprehensive understanding of the great variety of micro-organisms which play a part in the etiology of the disease. The internist or roentgenologist who enthusiastically administers *x*-ray therapy apparently does not fully appreciate the

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pathological physiology, not only of the ectatic bronchi but also of the related diseased lung parenchyma. The bronchoscopist who believes that weekly aspiration of the stem bronchi, with or without lavage with some "bactericidal" agent, is sound and adequate therapy, also fails to visualize the pathology, pathological physiology and bacteriology of the disease, as well as the limitations of bronchoscopic aspiration and "bactericidal" solutions. The following case serves to illustrate:

G. L., male, aged thirty-four (Figs. 12 to 15) was first seen in our clinic in 1934. He gave a history of having had bronchiectasis for seventeen years, and stated that during the preceding fourteen years, he had been bronchoscoped "more than 200 times." His willingness or apparent desire to continue with a method of treatment which apparently had had no beneficial influence on his symptoms, was probably because of the "reaction" which resulted from the large amount of cocaine used for the local anesthesia before bronchoscopy. The patient died of bronchopneumonia in 1938, having had saccular bronchiectasis for at least twenty-one years, and having been bronchoscoped something over 200 times, and having refused lobectomy because he "believed" bronchoscopy would eventually cure his disease.

It would seem that the time has come when sufficient knowledge is available regarding the fundamentals and the natural course of the disease so that a more unified opinion among the various specialties and specialists might crystalize as regards the treatment of bronchiectasis.

Although the present paper is especially concerned with the morbidity and mortality of medically treated bronchiectasis, some discussion of the present concepts of etiology, pathogenesis, and bacteriology, and certain principles of treatment based on these fundamentals, seems desirable. Sound management and treatment of bronchiectasis is dependent upon a comprehensive understanding of these fundamental principles, the various clinical patterns or types of the disease, its natural course, its obvious and potential hazards, the limitations of medical therapy and the curability of single and multiple lobectomy or pneumonectomy, and the morbidity and mortality of untreated and medically and surgically treated bronchiectasis.

ETIOLOGY AND PATHOGENESIS

Numerous theories and opinions have been advanced regarding the etiologic and pathogenetic factors of bronchiectasis. Not a few writers have emphasized the congenital nature of the condition and have theorized that the condition is not discovered more often earlier in infancy and childhood, because until infection results, the patient may not have symptoms to focus the attention of the physician on the congenital defect of the bronchi and lung.

Andrus and others have emphasized the importance of physical forces resulting from collapse of the lung in the production of bronchiectasis. Pediatricians, including Anspach, McNeil, and Richards have pointed out that collapse of a lobe may occur in children with pneumonia, and bronchiectasis is sometimes a sequel. Perry and King discuss the importance of "collapse" of a lobe following pneumonia as an important etiologic factor in their large series of cases, and state that, "in 167 patients (42 per cent of their series), the age of onset was in the first decade, a period when pneumonia, measles, whooping cough and other contagious fevers are common and these diseases may give rise to collapse."

Clerf, Jackson and others have discussed the importance of inhalation of foreign bodies with subsequent chronic pulmonary and bronchial infection, as an important etiologic factor in bronchiectasis.

Until a few years ago, most authors and teachers in this field held that bronchiectasis was secondary to chronic disease of the paranasal sinuses. Despite the fact that for many years this idea was almost universally accepted, considerable evidence has come to light recently to indicate that the disease is not necessarily always associated with or secondary to chronic sinus disease.

Goodale, Perry and King, have suggested that while bronchiectasis and chronic sinus disease are often associated, bronchiectasis is not always secondary to the former condition, nor is sinus disease always present in cases of bronchiectasis.

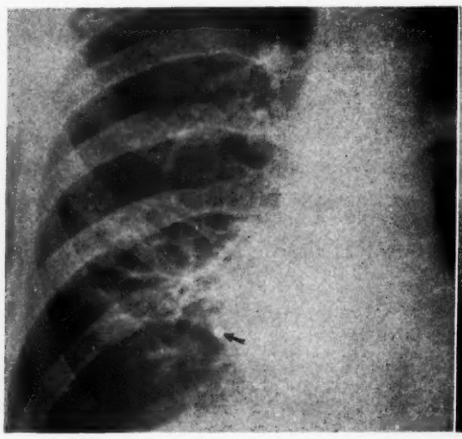


FIG. 1.

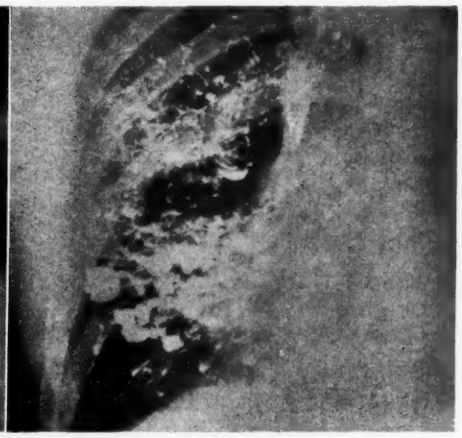


FIG. 2.

FIGS. 1 AND 2. W. B., white male, aged thirty-one, was first seen in 1937 complaining of cough, expectoration and hemoptyses. His parents had told him that he had "pneumonia" after "swallowing" a "buckshot" at the age of one. He had had cough and expectoration of mucoid to mucopurulent sputum as long as he could remember, was subject to chest colds and had had several attacks of "pneumonia" always on the right side. He had small recurrent hemoptyses during the past four or five years. When first seen, he was in good general health. There was marked clubbing of the digits and numerous sibilant râles and rhonchi were heard below the third anterior rib and seventh spine on the right. *X*-ray (Fig. 1) shows a foreign body, apparently a buckshot, at the level of the fourth anterior interspace right, and saccular bronchiectasis with a moderate amount of fibrosis. Bronchogram (Fig. 2) confirmed the diagnosis of extensive saccular bronchiectasis of the right middle and right lower lobes. Dr. Girard Oberrender was unable to see or remove the foreign body bronchoscopically. The patient refused lobectomy, is still in good general health and working full time.

In one hundred cases studied at the Lenox Hill Hospital during the past ten years, thirty or 30 per cent had definite chronic sinus disease. A careful analysis of the case histories of these thirty patients revealed that twenty or 66.6 per cent had symptoms, findings, or both of bronchiectasis, from several months to several years *before* they developed evidence of chronic sinus disease. From these observations and others made at Bellevue Hospital, some with Dr. Edith Lincoln on the Pediatric Service, and others on the Tuberculosis Service, a definite impression has resulted, that chronic sinus disease is perhaps more often *secondary* to bronchiectasis than it is a cause of, or important etiologic factor in, the development of bronchiectasis. In considering the etiologic relationship of the two conditions, it should be remembered that bronchiectasis usually has its onset during childhood or early adult life while chronic sinus disease has its onset relatively *later* in life. In their large series of cases, Perry and King reported that the onset of bronchiectasis occurred during the first decade in 42 per cent, and during the second decade in 27 per cent of their patients. They did not give figures to indicate the seniority relationship of the two diseases. In the series of cases studied at the Lenox Hill Hospital Clinic, the onset of the bronchiectasis apparently occurred in the first decade in 41 per cent and during the second decade in 23 per cent of the cases. These figures tend to confirm our belief that paranasal sinus disease is not infrequently secondary to bronchiectasis.

In the classical case of bronchiectasis which frequently develops following bronchopneumonia in children, often when streptococci are implicated, the early

pathological changes which eventually lead to demonstrable ectasia of the bronchi are not confined to the bronchial wall, but also

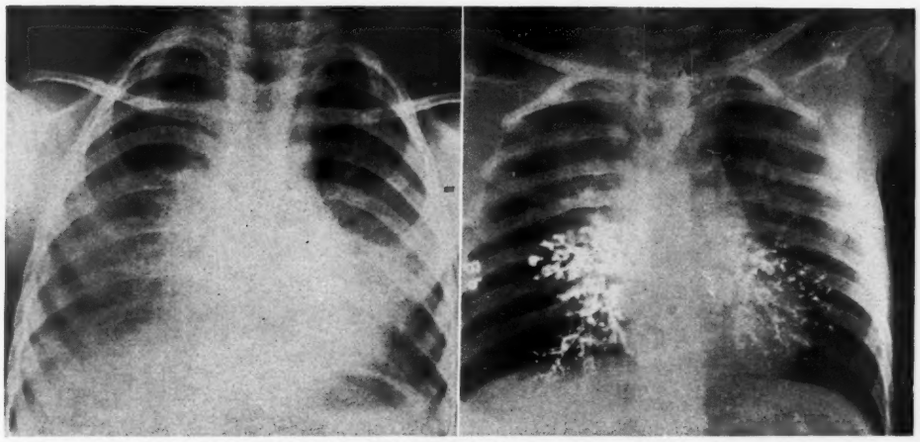


FIG. 3. FIG. 4.

FIGS. 3 AND 4. B. P., white male child, aged two when first admitted to the hospital in 1932 with bilateral bronchopneumonia which developed following measles. (Fig. 3.) On admission, the patient was acutely ill, cyanotic, dyspneic and required oxygen for several days. Dullness, diminished breath sounds and later, bronchovesicular and bronchial breathing and numerous râles were present over the middle and both lower lobes. Sputum typing was negative for pneumococci, but cultures showed hemolytic streptococci predominating. The patient made a slow convalescence. During the following winter, the patient had two attacks of bronchopneumonia with slow convalescences. Numerous moderately coarse râles persisted over the middle and both lower lobes. Lipiodol bronchogram shows the presence of early predominantly saccular bronchiectasis especially in the right lung. (Fig. 4.)

often, if not almost always, simultaneously involve the lung parenchyma. (Figs. 3 and 4.) Consequently, as a result of repeated attacks of bronchopneumonia, which may occur during a single winter and subsequent spring season, these rather insidious and chronic inflammatory changes in the bronchial mucosa and submucosa and lung parenchyma develop and progress. Serial chest roentgenograms at this time may show failure of the lungs to clear completely following one of these attacks of more or less atypical bronchopneumonia. During the summer season, the patient may improve clinically and roentgenological study may show almost complete clearing of the patchy and linear areas of residual inflammatory changes. However, during the following winter season, upper respiratory infections are apt to be followed by evidence of low-grade bronchopneumonia with associated chronic inflammatory changes of the bronchi. Often these chronic inflammatory changes fail to undergo complete resolution and healing and many of the areas progress to a state of irreversible fibrosis; and if the chronic infection continues or is repeated, as is likely, further fibrosis of the involved lobe with shrinkage and contraction of the lobe or segment, eventually results. Simultaneous with the development of these chronic parenchymal changes, similar progressive chronic inflammatory changes result in the bronchial wall. After prolonged or protracted periods of infection and repeated exacerbations, certain irreversible changes occur in the tissues comprising the bronchial wall. Areas of chronically infected granulation tissue replace the normal epithelial lining of the bronchus, the elastic fibers and the muscle bundles are partly or largely replaced with fibrosis, and in some instances, destruction of the cartilaginous structures of certain bronchi may result. As a consequence of these destructive onslaughts, weakening of the bronchial wall results. At this time, various physical phenomena, including shrinkage and contraction of a segment or of an entire lobe as a result of extensive fibrosis and, comparatively ineffective and

excessive coughing, combine with other factors to cause dilatation, or ectasia of the bronchi. Also, as a result of chronic inflammation, stenosis may develop in the stem, secondary or tertiary bronchi, frequently beyond the visualization of the bronchoscopist. As a partial consequence of the stenosis, the diseased and weakened bronchi become further dilated distal to the stenosis. Pooling of purulent and often anaërobically infected secretions occurs in the ectatic areas. These factors combine to bring about the saccular bronchiectasis often spoken of as congenital or cystic bronchiectasis.

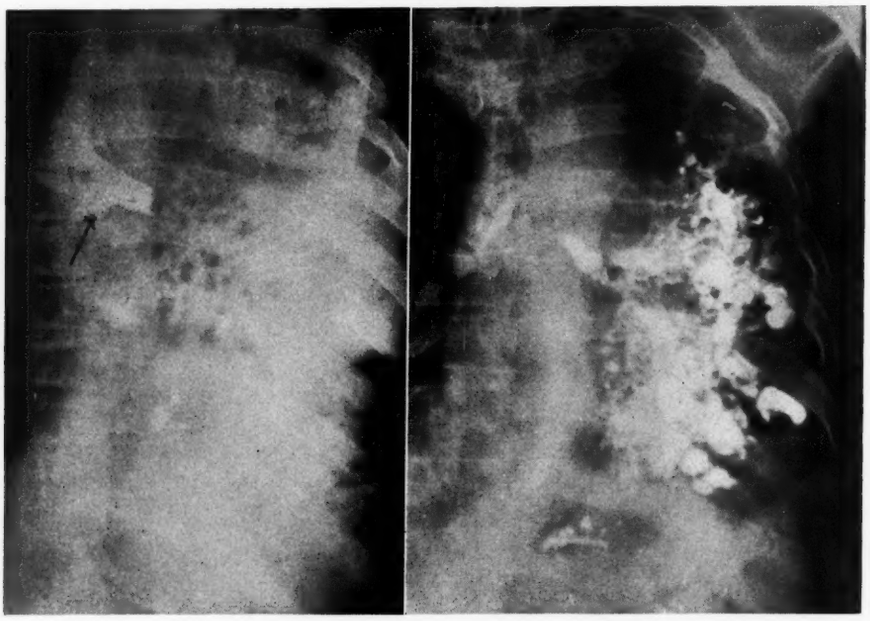


Fig. 5. Fig. 6.

Figs. 5 and 6. G. C., white male, aged fifty-seven, was admitted to the clinic in good general health with a history of chronic cough and small amount of expectoration for many years. Examination revealed dullness and absent breath sounds throughout the left chest. *X*-ray examination showed dense clouding throughout the left side with the mediastinal structures deviated to the left, and numerous annular areas of decreased density. Lipiodol bronchogram shows occlusion of the left stem bronchus. (Fig. 5.) Bronchoscopy revealed a hard, round tumor occluding the left stem bronchus, which was removed by Dr. John D. Kernan. Histologic studies showed the tumor to be osteochondroma. Following the removal of the tumor, the patient expectorated large quantities of purulent sputum having a foul odor. Bronchogram was repeated and revealed extensive saccular bronchiectasis involving most of the left lung. (Fig. 6.)

As a result of these extensive pathological changes in the lung parenchyma and bronchi, the normal cleansing power and the physiological function of the bronchi and lung become greatly impaired. Thus a vicious cycle develops which increases the vulnerability of the diseased lung and bronchi to further endogenous or exogenous infection. These changes make it obvious why many patients with bronchiectasis are likely to suffer protracted and repeated relapses and exacerbations, and also indicate why the disease is usually progressive, if not in the actual development of new bronchiectatic areas in previously healthy parts of the lung, certainly often in the development of an increasing amount of secondary or associated pulmonary fibrosis, emphysema, and/or suppurative pneumonitis.

Various other conditions may play a part in the development of bronchiectasis. Any condition which predisposes to, or causes the development of chronic inflammatory changes in the lung parenchyma and bronchi, may lead to the development of bronchiectasis. This is especially true in

FIG. 7.

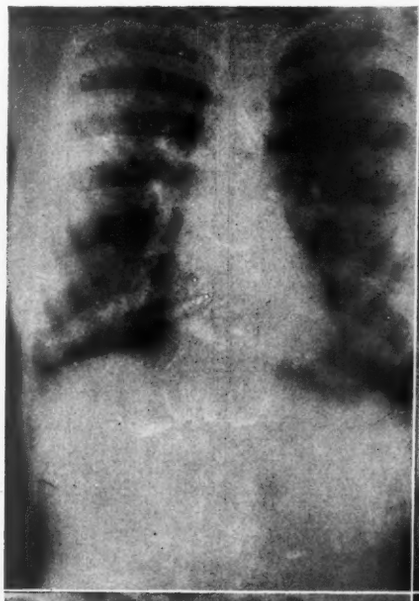
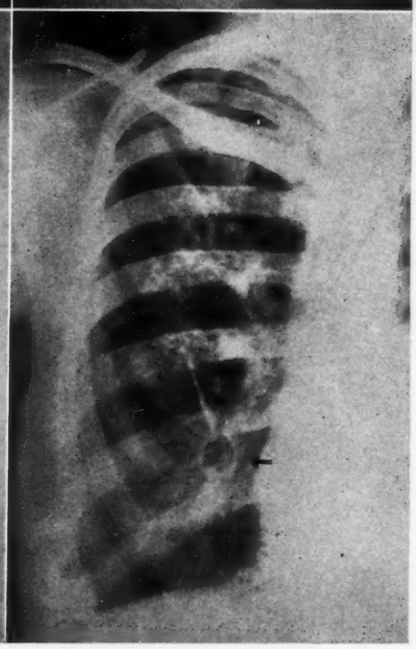


FIG. 8.

FIG. 9.

FIG. 10.



FIGS. 7 TO 10. H. G., white female, was admitted to the clinic in 1930 at the age of seventeen, with a history of chronic cough and expectoration, dyspnea, and repeated attacks of "bronchitis" and bronchopneumonia since early childhood. Examination showed an emaciated, tall girl, appearing chronically ill. There was no clinical evidence of chronic sinus disease and *x*-ray examinations of the paranasal sinuses were negative. The percussion note over both lungs was hyperresonant especially on the left, posteriorly. Breath sounds were diminished over both lung fields and numerous sibilant râles and rhonchi were heard over the lower half of both lungs. Chest *x*-ray (Fig. 7) reveals linear and patchy areas of fibrosis in the right lower two-thirds, and to a much less extent in the lower half of the left lung. Lipiodol bronchogram four years later (Fig. 8) shows cylindrical bronchiectasis involving the right middle and both lower lobes. At this time, the evidence of emphysema was much more marked than in 1930. The left diaphragm is markedly depressed. (Fig. 8.) Cough, expectoration and increasing dyspnea continued and the patient developed abscess in the right lower lobe. (Fig. 9.) The lung abscess was thought to result from a "spill-over" of infectious secretions from the bronchiectasis. Because of the poor general condition and marked dyspnea, surgical drainage was not done. During the next few months, the lung abscess gradually contracted (Fig. 10), and finally disappeared. Five years later, the patient is in fairly good health but remains quite dyspneic. This case illustrates two complications of bronchiectasis: (1) the development of marked emphysema, especially of the left lung (Fig. 8), and (2) lung abscess of the right lower lobe. (Fig. 9.)

children whose bronchi are more vulnerable to potential destructive forces than are bronchi in adults. Bronchial polyps, benign tumors, stenosis of the bronchi resulting from tuberculosis of the bronchial wall, or other causes, may interfere with the normal cleansing power of the bronchi and promote aspiration and retention of infectious secretions with the subsequent development of lung abscess, suppurative pneumonitis or indurative pneumonia and bronchiectasis. Partial or complete occlusion of relatively easily compressible bronchi of children, by enlarged caseous tuberculous tracheobronchial and mediastinal nodes, may not infrequently lead to atelectasis, chronic suppurative pneumonitis, indurative pneumonia and bronchiectasis. This mechanism is probably more often implicated in the development of bronchiectasis especially in children, than is generally recognized.

ASSOCIATED AND RELATED CHANGES IN THE LUNG PARENCHYMA

The gross pathological changes in the lung parenchyma in advanced cases of bronchiectasis may be proliferative or fibrotic, emphysematous and, destructive (cavities).

Patients with bronchiectasis rarely develop secondary lung abscess cavities. However, when well defined lung abscesses develop, they are usually located in previously "healthy" and nonbronchiectatic areas of the lung. The development of lung abscess in previously healthy areas of the lung undoubtedly come about as a result of a "spill over," or inhalation of infectious secretions originating in the bronchiectatic areas. While well developed saccular bronchiectasis has not been seen to disappear, we have observed the slow contraction and final disappearance of lung abscess cavities in cases with bronchiectasis. (Figs. 7 to 10.)

Proliferative or fibrotic changes in the lung parenchyma are present to a varying degree in most, if not all cases of bronchiectasis. The degree of such changes may be minimal and largely linear and scarcely demonstrable roentgenologically, occasionally even in cases with extensive bronchiectasis. The degree of pulmonary fibrosis is apparently often related to the amount and character of infectious secretions present. This relationship may also be just the reverse. We have not infrequently observed cases with extensive dry bronchiectasis in which fibrotic changes were conspicuous by their absence, to the extent that even saccular bronchiectasis might be overlooked on the roentgenogram.

In other cases in which there is considerable widely distributed cylindrical bronchiectasis of long standing, associated emphysematous changes may largely obscure roentgenological evidence of pulmonary fibrosis. In still other cases, emphysematous changes may so dominate the clinical and roentgenological picture, that the primary underlying bronchiectasis may not be suspected. Occasionally, the emphysematous changes may be largely limited to the bronchiectatic lobe or lobes. (Fig. 8.) This is particularly true in the earlier phases of the disease, but as lobar emphysema progresses, as is frequently likely, compensatory emphysematous changes now become obvious in the nonbronchiectatic lobes as well.

More or less simultaneous with the extensive development of pulmonary fibrosis and emphysema, secondary related changes of the thoracic cage gradually develop, further limiting the respiratory function and increasing the miserable existence of these sufferers of chronic anoxemia and toxemia. The intercostal spaces become widened, the entire bony cage is more or less fixed in the position of deep inspiration, the diaphragms are flattened and greatly depressed into the abdominal cavity and their bellows action is markedly diminished. As a consequence of these associated and related pulmonary and chest wall changes, the pulmonary reserve is greatly diminished and in many cases, may become so depleted that the least physical effort or slight upper respiratory infection may lead to moderate or

Fig. 11. R. F., a white female, was thirteen years of age when admitted to Bellevue Hospital in 1931. She was sent to the hospital because of an hemoptysis and with a diagnosis of advanced tuberculosis of the left lung. She gave a history of having had pneumonia "many times" during early childhood and had had cough and expectoration as long as she could remember. Examination showed a well nourished girl, apparently in good general health, and moderate clubbing of the fingers. The right lung was negative on physical examination but there was marked dullness and diminished to almost absent breath sounds throughout the left side. *X*-ray at this time showed dense homogeneous clouding throughout the left lung with small areas of decreased densities suggesting underlying bronchiectasis. The sputum was repeatedly negative for tubercle bacilli. Lipiodol bronchography revealed complete occlusion of the left main bronchus. Bronchoscopy revealed a tumor mass in the left main bronchus. A considerable portion of the tumor was removed and immediately thereafter the patient raised large quantities of purulent sputum having an extremely foul odor. The biopsy from the bronchus revealed benign adenoma, thought by some, however, to be carcinoma. The remainder of the bronchial tumor was removed through the bronchoscope by Dr. John D. Kernan. Serial *x*-rays showed marked clearing of the left lung. Lipiodol bronchogram (Fig. 11) shows extensive cylindrical and saccular bronchiectasis involving both lobes. The patient gradually lost all cough and expectoration, her general condition improved, and during the past ten years, she has completed her high school education and is now working full time as a stenographer, enjoying good health. The bronchiectasis has remained "dry" during the past seven years with rare exceptions following colds.

severe dyspnea or orthopnea, marked fatigue, weakness, mental lethargy, headache, anorexia, loss of weight and, not infrequently, marked psychological disturbances characterized by periods of depression, melancholia and, rarely, dementia. Blood studies may show compensatory pseudopolycythemia and hypochromic anemia. Such cases are hopelessly incapacitated, though frequently not actually ill or febrile for long periods of time.

As a result of the protracted anoxemia and toxemia, serious damage to many vital organs may eventually develop. Amyloid degeneration of the kidneys, liver, spleen and intestines is not infrequent. Myocardial degeneration, cor pulmonale, and eventually right-sided heart failure, and occasional brain abscess, may develop.

Bearing in mind the variety, degree and severity of the pathological changes both in the lung parenchyma and bronchi, and the frequency of anaërobic infection, often resulting in foul odor of the sputum, it is not difficult to visualize the morbidity and mortality of untreated or unsuccessfully treated cases of bronchiectasis.

MORBIDITY

While the mortality of untreated, medically and surgically treated bronchiectasis has been studied and reported by many clinics, the morbidity, a condition most difficult to appraise, has seldom been given the consideration it rightfully deserves.

Chronic invalidism of a physical and psychological nature, often from early childhood through a prolonged miserable life, with the ultimate prospect of death either resulting from pneumonia, lung

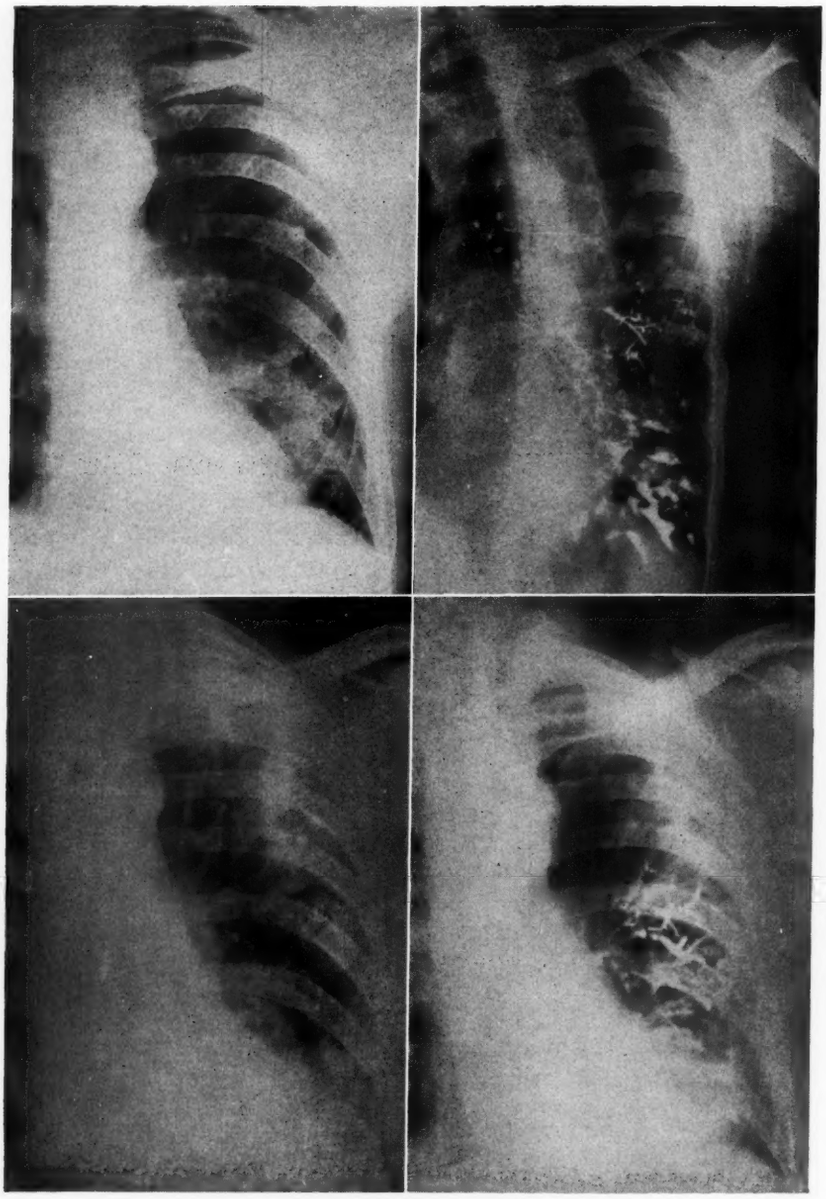


FIG. 12.

FIG. 13.

FIG. 14.

FIG. 15.

FIGS. 12 TO 15. G. L., a white male, was thirty-four years of age when admitted to the Lenox Hill Hospital Chest Clinic in 1934, with a history of chronic cough and expectoration, frequently with foul odor, since the age of seventeen. The cough and expectoration followed an attack of pneumonia on the left side at the age of seventeen. The patient was well nourished and appeared in good general health. There was marked clubbing of the fingers, and clinical as well as *x*-ray evidence of chronic sinusitis. Examination of the chest revealed dullness over the lower part of the left lower lobe, numerous coarse râles below the fourth anterior rib and eighth spine. Chest *x*-ray (Fig. 12) reveals clouding involving most of the left lower third. The patient had been bronchoscoped periodically for the preceding eleven years, however, apparently without any beneficial effect. Bronchogram reveals extensive saccular bronchiectasis of the lower portion of the left lower lobe. (Fig. 13.) Two series of deep *x*-ray therapy were given in the spring and summer of 1934. A total of 6,000 r units were given. Some of the treatments were mistakenly directed over the upper third of the left lung and following this, the patient developed pain, increased cough, expectoration and fever. *X*-ray examination at this time (Fig. 14) reveals dense clouding just below the clavicle on the left and perhaps slightly increased clouding at the base. The condition at the left top was diagnosed as *x*-ray pneumonitis and *x*-ray therapy was discontinued. The clouding gradually diminished but was still present almost two years later, and appeared to be largely fibrotic in nature at the time. (Fig. 15.) The bronchogram at this time revealed essentially no change in the condition of the bronchiectasis. The patient died of pneumonia several months later. This case illustrates the failure of repeated bronchoscopies and bronchoscopic lavage to influence the natural course of the disease and the disadvantages and dangers of *x*-ray therapy.

abscess, pulmonary hemorrhage, heart failure or some related complication, is the picture that both surgeon and internist should visualize in considering the morbidity and treatment of many patients with bronchiectasis. Unfortunately, the complete picture of the disease is not always visualized by the surgeon or internist. I have seen experienced general surgeons refuse to do lobectomy on a young nurse, in fairly good general health, with right lower and middle lobe saccular bronchiectasis, who had

Fig. 16. H. V., a white male, aged forty-two, was referred by Dr. Jacobsen in 1937 because of repeated hemoptyses, cough, large amounts of occasionally foul smelling sputum, chest pain, occasional bouts of fever and marked fatigue. *X*-ray examination of the chest in 1922 by another physician, revealed a large area of dense clouding in the mid-third of the right lung field adjacent to the hilum. The only symptoms at the time were severe chest pain and dry cough. Tumor of the mediastinum or right lung was diagnosed at the time (1922). Diagnosis was confirmed by several physicians and the patient was given large doses of deep *x*-ray therapy during the next few years by different physicians. Chest *x*-ray in 1937 showed a markedly shrunken and fibrotic right lung, apparently resulting from damage to the lung by large doses of *x*-ray resulting in chronic interstitial and indurative pneumonitis. Lipiodol bronchogram revealed extensive cylindrical bronchiectasis on the right. About one year later, January, 1938, the patient died of bronchopneumonia of the left lung. Autopsy by Dr. Jacobsen revealed, among other things, extensive chronic interstitial pneumonitis of the right lung with adhesive pleuritis, bilateral bronchopneumonia and extensive bronchiectasis of the right lung. The case is cited to show the deleterious effects on the lung tissue and bronchi of large doses of deep *x*-ray therapy. Figure 16 is a sagittal section through the right lung showing extensive fibrosis and thickened pleura and bronchiectasis. Courtesy of Dr. Victor Jacobsen, Troy, New York.

suffered a long period of invalidism, including social maladjustment and serious economic reverses, many of her patients not desiring her services because of chronic cough, severe halitosis and foul odor to the sputum, because at the time the patient was seen by the surgeons, the symptoms were comparatively mild—the optimum time for lobectomy.

In considering the morbidity of their living cases of medically treated bronchiectasis, Perry and King state that "most of the patients continue to have the same symptoms of cough, sputum and occasional hemoptysis. Often the amount of sputum increases and becomes foul in odor, though the patient mercifully may lose his appreciation of this fact through the loss of his sense of smell. He remains, however, unpleasant to relatives and friends, and suicide figuring twice in the causes of death in our series, indicates the patient's own unhappiness in living with his disease."

In assessing the "living capacity" of the nonsurgically treated group of their patients, the same authors estimated that "38 per cent were in excellent condition (despite the fact that more than 90 per cent continued to have cough and expectoration, and almost a third continued to have occasional hemoptyses), 42 per cent were in fair condition and 15 per cent in poor condition, with 5 per cent in extremely poor condition."

Despite these rather high percentages of patients in excellent to fair condition in this exceptionally well studied and large series of cases, it is well known that the condition of bronchiectasis patients ebbs and flows with the years and the seasons. In this connection, it might be mentioned that, while we agree in part with Perry and King that bronchiectasis, *per se*, rarely spreads to lobes other than those involved when the patient is first seen, nevertheless, we have frequently observed areas of fibrosis and suppurative pneumonitis enlarge and extend to previously uninvolved lung, and associated emphysematous changes become progressively worse. These fibrotic and emphysematous changes not infrequently dominate the clinical picture. Since relapse and remission, progressive fibrosis or pneumonitis, emphysema, chronic toxemia and anoxemia, characterize the natural course of the disease, it is reasonable to assume that the observed morbidity and mortality of bronchiectasis become greater the longer the cases are under observation.

Estimating the morbidity of any group of chronically ill patients is difficult and hazardous, and at best, founded on impressions rather than scientific facts. Despite these handicaps, an appraisal of the one hundred cases of bronchiectasis observed at the Lenox Hill Hospital Clinic during the past ten years, has been attempted by studying the number of hospital admissions, the frequency and severity of respiratory infections, the attacks of bronchopneumonia and acute or subacute attacks of so-called bronchitis, and pleurisy, the occurrence of hemoptyses, miscellaneous related illnesses, chronic sinus disease, the ability of the patient to work, their psychological state and social adaptability, and finally, whether or not the disease remained unchanged, improved or became worse. While a statistical analysis of these data is impossible, nevertheless, a fairly definite impression based upon close and prolonged personal observation and supplemented by a detailed analysis, give one a fairly complete picture of the morbidity in this group of cases.

Readmission of many of these patients to the hospital ward for reasons related to their bronchiectasis, was generally quite frequent, and varied from one to seven times during the maximum observation period of ten years. The definite impression is gained that readmission to the hospital of our bronchiectasic cases, was considerably more frequent than was true of patients having pulmonary tuberculosis and treated in the same clinic. Respiratory infections, including so-called bronchitis, atypical and typical attacks of bronchopneumonia, occurred much more frequently in the group of bronchiectasic patients than

in the patients with tuberculosis. Many bronchiectasic patients had from three to five atypical or typical attacks of bronchopneumonia during the ten-year period. It is reasonable to assume, that others had mild attacks of bronchopneumonia without being admitted to the hospital. Hemoptysis, though usually not large in amount, was a frequent and disturbing symptom, and was often followed by febrile attacks, with increase in cough and expectoration and occasionally the development of foul odor to the sputum. So-called head colds, exacerbation of chronic sinusitis, pleurisy, and asthmatic and asthmatoid attacks occurred frequently and seriously interfered with their work or studies in school.

Both clinical and laboratory studies show that many of these patients are "under-par" and not uncommonly develop nonpulmonary conditions directly or indirectly related to their poor general health. It was of interest that one patient developed complete prolapse of the rectum, and several others, inguinal herniae, conditions apparently indirectly related to chronic and excessive cough.

The working ability or the desire to work on the part of many of these patients of this economic status, is difficult to evaluate with any degree of accuracy, especially when present day relief measures often provide these people with greater economic security than does the work which is available to them, and which is frequently interrupted during the winter and spring seasons by exacerbations of their symptoms or relapse of their disease. In the living and traced patients, approximately 25 per cent have been able to do full time work as a general rule, and 40 per cent have been able to do part time work, their work being frequently interrupted by relapse of the disease. The remaining 35 per cent were either unable to work at all because of the severity of their symptoms, or had given up the idea of economic rehabilitation because of the psychological effect of the disease. These various physical and psychological factors which constitute the morbidity of the disease are undoubtedly responsible for the frequent failures in most of their endeavors of life. Little wonder that they become frustrated, depressed and are not over energetic or ambitious.

Churchill has forcefully emphasized the psychological handicap or morbidity of bronchiectasis. An appraisal of our patients in this regard reveals that this very important handicap was seldom properly evaluated by the physician and was frequently largely left to the social worker in the clinic or relief agency. In many patients, serious psychological handicaps undoubtedly play an important part in their social maladjustments, more especially those having chronic cough and foul odor to the sputum. The fact that 70 per cent of our adults are still single, and 6.6 per cent of those married are either divorced or separated, and that only 25 per cent are doing full time work, many of these their own housework, emphasizes the great importance of the psychological as well as physical morbidity which apparently caused three of our patients to threaten suicide. Seemingly it also was an important factor in the development of paranoia in another patient, necessitating hospitalization in a psychiatric institute, and led two of Perry and King's patients to commit suicide.

Thus, chronic invalidism beginning in childhood and often continuing throughout life, characterized by mental lethargy and inaptitude, physical unfitness and debility, and social maladjustment, is not infrequently the inevitable picture of many advanced, untreated and unsuccessfully medically treated cases of bronchiectasis. Indeed the morbidity in many cases of advanced bronchiectasis with suppurative pneumonitis, consisting of chronic invalidism, psychological changes ranging from mild depressive states to psychopathic personalities, complete economic insecurity, a life alone, apart, helpless and hopeless, is perhaps a greater problem for the internist, psychiatrist and surgeon than is the actual mortality of bronchiectasis, convincing as it is of the ultimate fate of

most patients with untreated and medically treated bronchiectasis. Little wonder that Churchill states that young adult patients rarely hesitate to elect a chance for cure if the possibility is offered. On the contrary, we regret with Churchill, that "Unfortunately, we still see the patient who returns to his family doctor for advice and is told that the operation (lobectomy) is an impossible one and that to consent to it means certain death."

An estimation of the present status of our untreated and medically treated living cases based on their symptoms, general and local conditions, shows that 13.3 per cent are regarded as improved, 46.6 per cent essentially unchanged and 41.1 per cent definitely worse than when first seen. We have not succeeded in curing any case of well developed bronchiectasis with medical measures including climatic treatment, *x*-ray therapy and bronchoscopy. The treatment of bronchiectasis is surgical, where major surgery is feasible and not contraindicated.

MORTALITY OF UNTREATED AND MEDICALLY TREATED BRONCHIECTASIS

While it is well known that many patients with extensive bronchiectasis may live out their normal span of life, it is also well recognized that this is an exception to the general rule. From reports in the literature and observations at Bellevue and Lenox Hill Hospitals, it is the impression that the majority of patients with bronchiectasis, succumb to some form of respiratory illness, or complication, either before or during the fourth or fifth decades.

Comparatively few large series of cases have been followed for sufficiently long periods of time, and for this reason, available comprehensive statistical studies of the mortality of the disease are somewhat limited. The reported mortality rates vary widely, depending upon the duration of observation, age at onset, type, extent, severity and location of the disease, and the predominant types of micro-organisms implicated. The prognosis in cases of relatively "dry" bronchiectasis involving the right upper or middle lobe with very little pulmonary fibrosis and emphysema, is relatively good in comparison with that of bilateral lower lobe saccular bronchiectasis with secondary anaërobic infection, extensive pulmonary fibrosis or suppurative pneumonitis.

In a group of forty-nine nonsurgically treated cases, observed for a period of six years, Roles and Todd report a mortality rate of twenty-three, or 47 per cent. This rate is considerably higher than that usually found in cases observed for similar periods of time. In Head's series of 200 cases, he found that of those having the onset during the first decade, but few were living after the age of forty. While our observations have been somewhat similar to those of Head, we have a considerable number of patients still living in their sixth and seventh decades, in whom the onset of bronchiectasis was in childhood.

G. E., male, aged seventy-six, has been under observation eleven years and gives a history suggesting the onset of his bronchiectasis at the age of five or six years. He has extensive bilateral cylindrical bronchiectasis involving both lower lobes with a moderate degree of pulmonary fibrosis and emphysema.

D. S., male, aged fifty-six, was under observation for eight years. He gave a history of repeated hemoptyses following "pneumonia" at the age of eight. When first seen ten years ago, he had saccular bronchiectasis involving the entire left lung with extensive pulmonary fibrosis resulting in marked displacement of the mediastinal structures to the left. Conventional serial chest roentgenograms over a period of twenty-one years, and lipiodol bronchography during a period of nine years, showed essentially no change of the pulmonary fibrosis or bronchiectasis. However, the patient finally succumbed to his pulmonary disease, dying of pneumonia at the age of sixty-four, after having had *known* saccular bronchiectasis for twenty-one years, and a history indicating that he had had bronchiectasis for a total of fifty-six years.

These isolated cases are of interest in that they indicate that occasionally pa-

tients with extensive bronchiectasis may live out the usual estimated life span, despite numerous relapses and exacerbations of the disease.

In their excellent paper on 400 cases of proved bronchiectasis, Perry and King report their findings regarding the natural course and prognosis of medically and surgically treated cases. They state that "A twelve-year study of the entire group showed the mortality in the nonsurgically treated cases 26 per cent, 41 per cent of these dying within five years of the onset and 15 per cent living twenty years or longer after the onset. Seventy-eight per cent of the dead died directly as the result of their disease." They state further that, "The working and living capacity of the traced living patients was considered to be excellent in 67 per cent of the surgical group and in 38 per cent of the nonsurgical group."

The estimated duration of the bronchiectasis of the entire series of cases (living and dead), observed at Lenox Hill Hospital, is 16.4 years. The average age of these patients is 35.8 years. The known mortality of the series collected during ten years with many being observed only for the past three or four years, is twelve or 12 per cent. However, fifteen patients or 15 per cent have not been traced during the past year and their status is unknown. The mortality for traced medically treated patients is 14.1 per cent.

TREATMENT

Remission and relapse, natural phenomena of the disease, should be borne in mind when attempting to evaluate the value of any form of therapy.

Since the etiology of bronchiectasis is dependent upon many factors, any attempt at prophylaxis must be on a general basis and essentially designed to build up the patient's resistance to infection and disease. The onset of bronchiectasis being most frequent during the first decade of life, the prevention of the disease devolves about the management of the usual diseases of childhood, especially the exanthemas, pneumonia, and more especially, streptococcal and influenzal pneumonia, and is primarily a problem of the pediatrician.

Children unduly susceptible to respiratory diseases, especially frequent colds, acute bronchitis and bronchopneumonia, should have more careful observation than is usual. This should include longer periods of bed rest than are customarily given to patients with upper respiratory infections, and every effort to improve the general condition and increase resistance to infection. In addition to the usual measures taken to bring about this result, removal of the child to a warm, dry climate during the winter and spring seasons may be of great value. Climatic treatment for the extensive case of bronchiectasis is only palliative at best and should never be substituted for surgical treatment where this type of treatment is indicated and feasible.

The prompt removal of foreign bodies (Figs. 1 and 2), polyps and benign tumors (Figs. 5 and 6), is of great importance, if serious infection, frequently anaërobic in nature, with the subsequent development of bronchiectasis is to be prevented. Prompt and adequate treatment of lung abscess, including surgical drainage, aids in the prevention of residual bronchiectasis. Although prophylactic treatment of bronchiectasis at best has very definite limitations, nevertheless, the prevention of bronchiectasis is probably less difficult than its cure. Insofar as our present knowledge goes, there is no medical cure of well developed bronchiectasis. The only known cure is surgical extirpation of the diseased part of the lung.

During the past number of years, we have tried the usual methods of medical treatment, however, without any worth while success except for symptomatic improvement. These measures consist of postural drainage, usually three or four times daily, beginning with the early morning, and repeating again just before retiring, the usual efforts to improve the general condition and increase the resistance to

infection, occasionally, pneumothorax in cases of relatively dry hemoptoic bronchiectasis to control hemorrhage, heliotherapy in the form of ultra-violet light to the entire body during winter, occasionally, autogenous vaccines, sometimes combined with cold vaccine, with the hope of preventing or minimizing upper respiratory infections and colds, thereby preventing or diminishing their ill effects on the bronchiectasis. We have not given arsenicals, but have had patients who transferred to our clinic, who had received arsenicals elsewhere, apparently without any beneficial effect whatever.

X-ray Therapy. In 1934, Berck first advocated *x*-ray therapy for the treatment of bronchiectasis. In his report of a case, he stated that "The purpose of this communication is to state the rationale and report the results of radiotherapy in the treatment of a case of bronchorrhea in bronchiectasis." He postulated that "The purpose of the application of radiotherapy, briefly, is to render the hypertrophic, hypersecreting bronchial mucosa atrophic and thus converted a 'wet' bronchiectasis into a 'dry' bronchiectasis."

The assumption by Berck that "the hypertrophic hypersecreting bronchial mucosa" is the source of the purulent secretions in bronchiectasis with suppurative pneumonitis seems highly theoretical and contrary to the general conception of the origin of purulent secretions in bronchiectasis. It seems more logical to assume that such secretions are the product of suppurative pneumonitis and ulcerative bronchitis and bronchiectasis rather than the result of a hypersecreting hypertrophic bronchial mucosa. Often, the bronchial mucosa no longer exists as such in bronchiectasis, but may be largely replaced by chronically infected granulation tissue. Berck's theoretical reasons for *x*-ray therapy for bronchiectasis seem illogical and not founded on the gross pathology and pathological physiology of the disease. By 1939, Berck and Harris had largely abandoned their first theory explaining the rationale of *x*-ray therapy for bronchiectasis and, at that time, offered a second theory. They state: "Further explanatory hypotheses may be adduced to explain the successful action of roentgen rays in suppurative bronchiectasis, such as, possible enhancement of immunity processes both through the action of the rays in stimulating antibody action, and the physico-chemical alterations of the local tissue reactions." They concluded in this report that roentgen therapy for suppurative bronchiectasis is both "feasible and successful."

Because of Berck's encouraging report of 1934, several patients with bronchiectasis were subjected to *x*-ray therapy. Most of the treated patients experienced mild to moderate exacerbation of their local pulmonary symptoms and some developed what appeared to be related febrile attacks while receiving treatment. Occasional patients also developed what appeared to be related temporary remission of symptoms following treatment. We have followed these patients with particular interest during the past few years and find that the natural course or behavior of their disease has been quite similar to that before *x*-ray treatment was begun. We have not observed lasting improvement in any case. On the contrary, we believe that this form of treatment may result in the development of further pneumonitis and irreversible fibrosis. The following case illustrates the potential and actual disadvantage and danger of roentgenotherapy in the treatment of bronchiectasis:

G. L., male, aged thirty-four, was admitted to the Lenox Hill Chest Clinic in 1934 with saccular bronchiectasis of the lower half of the left lower lobe, and cylindrical ectasia in the lingula of the left upper lobe. (Figs. 12 to 15.) Therapeutic bronchoscopy had been done over a period of many years before coming to our clinic, apparently without any improvement in his condition. Lobectomy was advised but refused. Roentgenotherapy was suggested and accepted. Before roentgenotherapy was begun, there was a moderate amount of fibrosis limited to the lower third of the left lung field. The

upper half of the left lung was normal on physical and roentgenological examinations. (Figs. 12 to 15.)

In the latter part of 1934, and in the summer of 1935, the patient was given two series of roentgen-ray treatments by the Radiological Department. Approximately 1,500 r were given through each of several portals, with a total of 6,000 r. Some of the treatments were inadvertently directed to the upper half or normal part of the left lung. A few days after receiving treatment over the upper half of the lung, the patient developed pain in the left upper chest, with a temperature rise to 101.6°F. and increased cough and expectoration. After admission to the hospital, the chest roentgenogram revealed a new area of clouding in the left upper half and also increased clouding in the left lower third. At first these changes were thought to be the result of bronchopneumonia. However, the persistence of the inflammatory changes in the left upper third, where there was no bronchiectasis, and their final conversion into fibrosis which persisted to some degree for over two years, or until the patient's death, convinced the staff that the condition was one of *x*-ray pneumonitis.

The fortuitous circumstance that resulted in the patient receiving considerable *x*-ray therapy over the left upper, or normal lung field, offered an opportunity to evaluate more accurately, the effect of such a dose of roentgenotherapy on a part of this patient's normal as well as diseased lung. Prior to this experience, evidence of increased pneumonitis, following *x*-ray therapy to bronchiectatic areas, was thought to be due to an exacerbation of the chronic pneumonitis. However, the above and other observations, and the work of Desjardins, Jacobsen and others, convinced us that roentgenotherapy, when the dose is sufficiently great and purposefully directed through several portals to converge on a selected area within the lung, may cause acute inflammation of the lung or pneumonitis. If the damage to the lung is sufficiently severe, permanent or irreversible fibrosis may result and actually aid in the development of bronchiectasis. This has been observed in a case reported by Jacobsen. (Fig. 16.)

Because roentgen therapy for bronchiectasis seems illogical, and having observed acute pneumonitis and irreversible fibrosis and emphysema develop in a considerable number of treated patients, and since such damage may lead to further bronchiectasis and adhesive pleuritis, making future thoracic surgery more difficult, it is believed that this method of treatment should be strongly discouraged.

Bronchoscopy. Diagnostic bronchoscopy is indicated in most cases with suspected or known bronchiectasis. However, it is most doubtful if therapeutic bronchoscopy is of any benefit except in a very limited number of patients, namely, those having foreign bodies in the bronchi, benign bronchial tumors, partial bronchial occlusion or stenosis of the stem bronchi.

Bronchoscopic lavage with various so-called "bactericidal" agents advocated by some bronchoscopists is not only useless but dangerous. Such treatment increases the amount and liquidity of infectious secretions and materially enhances aspiration of these secretions into relatively healthy areas of lung tissue. We have seen severe febrile reactions with the subsequent development of new areas of pneumonitis following bronchoscopic lavage. This type of treatment is useless and should be discouraged.

The failure of medical treatment of bronchiectasis, the protracted morbidity and high mortality, its tendency to localize in one or two lobes, have encouraged thoracic surgeons during the past number of years to perfect some successful method of treating the disease. Many recent reports of the successful surgical treatment of bronchiectasis and suppurative disease of the lung by Churchill, Overholt, Churchill and Beasley, Bohrer, Berry, O'Brien, Haight, Heuer, Walker and others, are convincing of the rapid advances made during the past few years in the surgical treatment of bronchiectasis.

In 1940, Perry and King reported that "the operative mortality in 122 modern type lobectomies performed on 116 patients

by Dr. Edward D. Churchill was 3.3 per cent." This exceedingly low mortality rate is convincing evidence that lobectomy for selected cases of bronchiectasis by experienced thoracic surgeons is not only a safe procedure but the best curative measure for bronchiectasis. Those who are content to continue with postural drainage, bronchoscopic lavage, vaccines, roentgenotherapy, climatic treatment, arsenicals, and are misled into believing that the natural temporary remission of symptoms is the result of these medical measures, cannot be aware of the natural behavior of the disease and the feasibility and curability of lobectomy in many cases of bronchiectasis.

CONCLUSIONS

1. Wide differences of opinion still exist regarding many aspects of bronchiectasis.

2. Congenital bronchiectasis is an extremely rare condition and is apparently frequently confused with acquired saccular bronchiectasis.

3. Bronchiectasis usually has its onset in childhood or early adult life. Chronic sinusitis usually has its onset relatively later in life.

4. Chronic disease of the paranasal sinuses is perhaps more often secondary to bronchiectasis than is bronchiectasis secondary to paranasal sinus disease.

5. There is no satisfactory medical treatment or cure for bronchiectasis. However, certain medical measures may aid symptomatic improvement.

6. The morbidity and mortality of untreated and medically treated bronchiectasis and suppurative pneumonitis is such that the physician who routinely advises young adults with operable bronchiectasis against surgery, is assuming a grave responsibility and in all probability renders his patient a great disservice.

7. Most patients with bronchiectasis and suppurative pneumonitis should be considered for lobectomy or pneumonectomy although many may be found inoperable.

8. Modern lobectomy by experienced thoracic surgeons is a safe and curative procedure for bronchiectasis.

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PULMONARY CYSTS*

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THE terms, "pulmonary cyst" and "cystic disease of the lung," have been applied to a wide variety of pathological processes in the lung. Some authors include in the category of cysts all sizeable fluid or air-containing cavities within the lung that are not primarily the result of destruction of pulmonary tissue by suppuration or caseation. Other authors use the term, "cystic disease of the lung," as synonymous with "congenital cystic disease," thereby inferring that all pulmonary cysts are congenital. Some cases that have been reported as examples of cystic disease of the lung, however, have been acquired lesions. As a result, there is much confusion in the literature concerning terminology, and authors are in disagreement as to what conditions should be included as pulmonary cysts.

The usage of the term, "pulmonary cyst," without qualification for a wide variety of intrapulmonary air-containing cavities is unsatisfactory. Although at the present time it is sometimes not possible to determine clinically the exact nature of the cavity present in a given case, attempts should be made to differentiate the various types on the basis of etiology and pathology. The mechanics of the cavity formation and the structural characteristics of the cavity wall determine the distinctive clinical features of the various types of cyst-like pulmonary conditions. Rational treatment can be applied only when the factors which differentiate the various types of lesions are evaluated.

The formation of a cavity in the lung depends on either (1) developmental abnormality, (2) destruction of pulmonary tissue by an inflammatory process, or (3) hyperinflation of a small defect in the pulmonary parenchyma, or a combination of these processes. Persistence of intrapulmonary cavities may be due to one or more of the following factors: (1) positive intracavitary pressure, (2) progressive destructive process within the lung, (3) loss of expansibility of surrounding pulmonary tissue, (4) elastic properties of the cavity wall, or (5) epithelization of the cavity wall.

The various factors just enumerated determine whether a given pulmonary cavity is likely to disappear spontaneously, whether the cavity can obliterate itself after drainage or whether excision of pulmonary tissue is necessary. A cavity which owes its size chiefly to hyperinflation by a check-valve mechanism of the communicating bronchus may have a different clinical course than a cavity produced by destruction of pulmonary tissue. Decompression or drainage may be all that is required for the disappearance of a nonepithelized cavity if the surrounding lung is elastic, but drainage alone will not result in obliteration of a cavity completely lined by epithelium even though the adjacent pulmonary tissue is normal.

The following types of lesions have been included under the term of pulmonary cyst by some authors:

I. *Nonparasitic Cysts and Cystlike Cavities:* 1. Congenital pulmonary cysts: congenital fluid or air-containing cavities in the lung which may or may not have an epithelial lining. The cysts may become expansile because of positive intracavitary pressure. The lesions are due to developmental defects or intrauterine disease, but secondary infection may be superimposed.

2. Cystic bronchiectasis: cyst-like dilatation of the bronchi which may be congenital or acquired.

3. Acquired intrapulmonary cavities originally produced by the destruction of

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pulmonary tissue. The cavity may or may not be epithelized.

4. Pneumatoceles: nonepithelized positive pressure cavities produced by the hyperinflation of a defect in the pulmonary parenchyma resulting from pulmonary infection.

Fig. 1. Large infected pulmonary cyst in a four-year old child which was treated as an empyema for two years. Cyst successfully removed after diagnosis was established by biopsy of cyst wall which revealed lining of stratified columnar epithelium.

5. Emphysematous bullae: nonepithelized pulmonary cavities produced chiefly by the disruption of interalveolar septa.

6. Pulmonary blebs: localized collections of air within the pulmonary interstitial tissue.

II. *Parasitic Cysts.*

CONGENITAL PULMONARY CYSTS

Various pulmonary lesions have been classified in the literature as congenital cysts of the lung or congenital cystic disease. In some such instances the congenital origin of the lesion is either open to question or the lesion is definitely an acquired one. It is often impossible to ascertain definitely on the basis of the history and a single roentgenogram whether the cystic disease is congenital or acquired. This applies to children as well as adults. A series of roentgenograms may aid in the solution of the problem by demonstrating changes which are inconsistent with one or the other type. In some instances the suspected congenital nature of the cyst has been disproved by the chance existence of a previous roentgenogram demonstrating no pulmonary abnormality.[10] It must be realized that infection and mechanical derangement in the lung may result in the formation of huge intrapulmonary air cavities. These pneumatoceles and emphysematous bullae frequently cannot be differentiated radiographically from congenital cysts of the lung. The problem is not merely of academic interest because the behavior of congenital and acquired cysts differs somewhat and the treatment may, therefore, be altered.

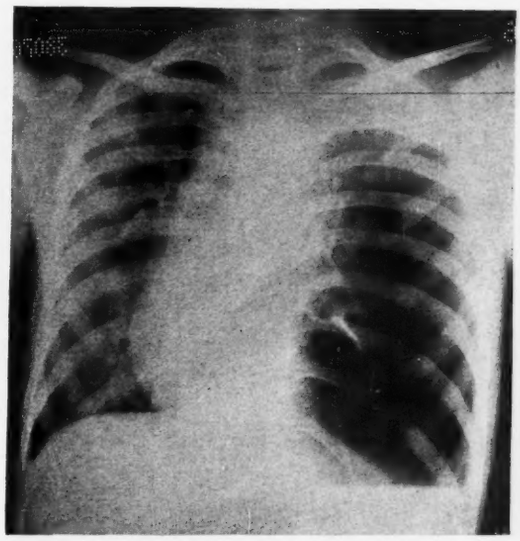


Fig. 2. Huge pulmonry cyst simulating a tension pyopneumothorax. Note strand of tissue extending out from pulmonary hilar region and traversing cavity. (Fig. 3.) There is considerable mediastinal displacement. Prolonged drainage resulted in little diminution in size of cavity.

Congenital cysts of the lung may be fluid or air-containing and may be single or multiple. The size of the cysts varies markedly. It has been assumed by some authors that all the air-containing cysts were originally fluid cysts which later ruptured into a bronchus. Following the discharge of the fluid contents, air entered the cyst cavity. The frequency of this sequence of events cannot be established on the

basis of the data available at present, but it seems to be an uncommon occurrence.

Congenital cysts may be lined by a cylindrical, cuboidal or flattened epithelium or the cyst may have no definite epithelial lining. The epithelium may be destroyed by secondary infection. Some of the cysts, but especially those without an epithelial lining, may become inflated to a huge size by a check-valve mechanism of the communicating bronchus.

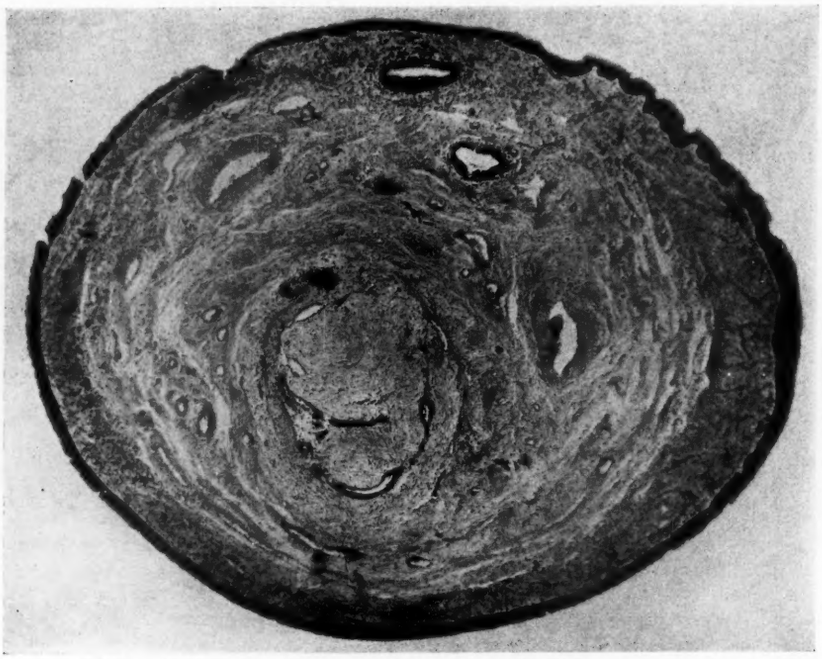


Fig. 3. Photomicrograph of cross-section of strand of tissue traversing cavity in Figure 2. There is a complete covering of stratified columnar epithelium and the cord-like strand contains bronchi. These findings indicate that cavity is intrapulmonary.

Koontz[8] in 1925 could find only 108 cases of congenital cysts of the lung in the literature. Included in this group were a few cases of conditions such as diverticula of the trachea or main bronchi, solitary cysts of the tracheal or bronchial wall, and cysts of aberrant lung tissue and accessory lobes.. Moreover, it is impossible to be sure that all of the pulmonary lesions were congenital in origin. The paper by Koontz stimulated interest in the subject and a large number of reports have appeared since.[13] In 1937, Schenck[12] collected 381 cases of congenital cystic disease of the lung from the literature, but the congenital nature of the cysts in some cases is open to question. Schenck[12] studied the incidence of the various types of cysts. In 38 per cent of the cases the cysts were solitary and in 62 per cent multiple. In 23 per cent of the cases solitary or multiple fluid cysts were present and in the other cases air cysts were found. Somewhat less than half of the cases were observed in infancy and childhood. Congenital cystic disease of the lung may be associated with other congenital anomalies. Cystic disease may be found in aberrant intrathoracic lung tissue.[5]

Congenital pulmonary cysts may cause symptoms shortly after birth or in childhood, but in some instances there are no pulmonary symptoms until adult life or the pulmonary cysts are unexpected roentgen or autopsy findings. The most common symptoms are cough, dyspnea and expectoration. Recurring attacks of dyspnea and cyanosis are common in infants. Fever, hemoptysis and thoracic pain may be present, especially in adults. Dyspnea and cyanosis are most prominent in the positive pressure air cysts.

The fluid-containing congenital pulmonary cysts usually cause no symptoms until secondary infection occurs. If the cyst is large or if there are multiple cysts, dyspnea may be present. When the fluid cyst ruptures into a bronchus, an air cyst may simply result,[1] or the cyst may become filled with pus. Infected solitary pulmonary cysts, whether congenital or acquired, may simulate a pulmonary abscess or an encapsulated empyema. (Fig. 1.)

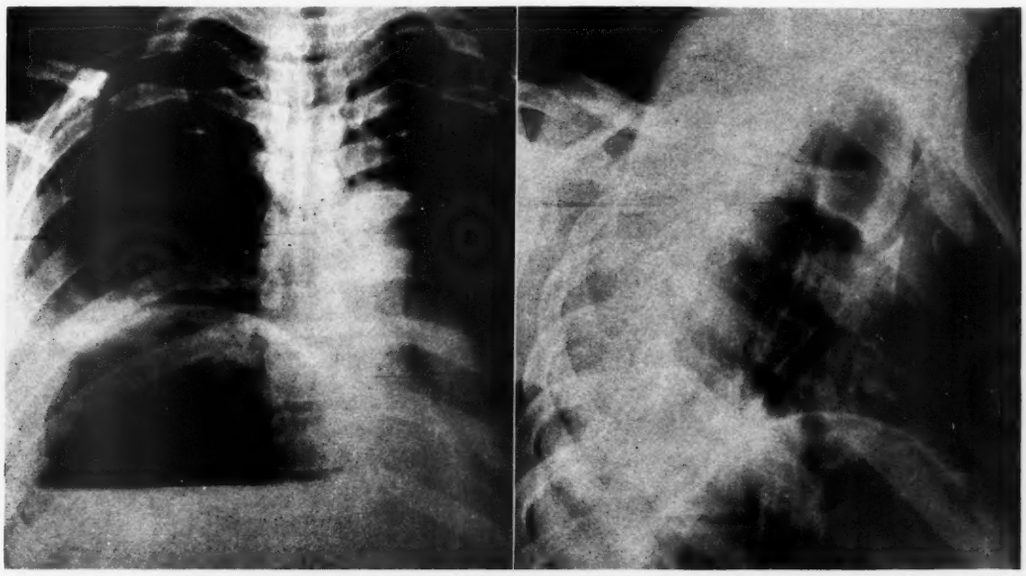


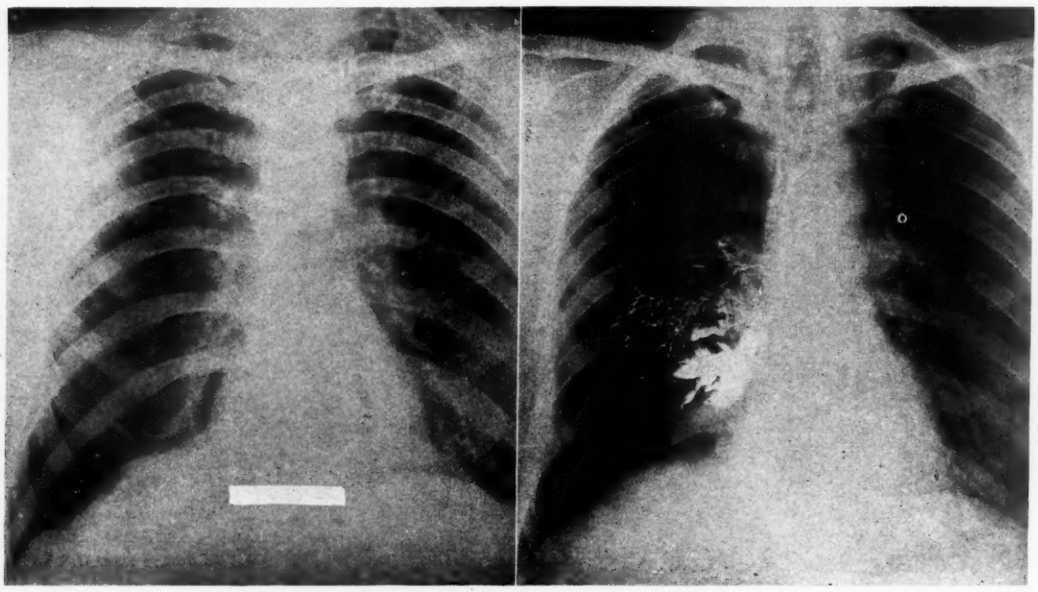
Fig. 4. Large infected intrapulmonary cavity in an adult. Following thoracotomy with drainage the cavity obliterated completely. Biopsy showed no epithelial lining.

Air cysts may be asymptomatic, but not infrequently due to a check-valve mechanism of the communicating bronchus the cyst becomes hyperinflated and may assume a huge size. These positive pressure cysts may compress the adjacent pulmonary tissue to a marked degree so that dyspnea and cyanosis may result. These balloon cysts may encroach on the remaining pulmonary tissue to a sufficient degree to cause death due to respiratory insufficiency.

The physical signs vary and are rarely diagnostic. The roentgenographic findings are the most important clinical evidence in the diagnosis of pulmonary cysts. The fluid cysts appear on the roentgenogram as round or oval areas of increased density. If the cyst communicates with a bronchus, a fluid level may be present. The air-filled cavities appear as round or oval areas of increased radiolucency. The location and contour of the area of radiolucency may serve to differentiate an intrapulmonary cavity from a localized pneumothorax. In expansile cysts there may be evidence of compression of the surrounding pulmonary parenchyma. The large positive pressure cysts may occupy almost an entire hemithorax and may even displace the mediastinum markedly toward the opposite side. (Fig. 2.) This latter type of cyst is frequently incorrectly diagnosed as a tension pneumothorax. The two conditions can usually be differentiated radiographically, however, because in the case of a huge cyst there is no prominence at the pulmonary hilum representing collapsed lung, and the costophrenic sinus contains compressed pulmonary tissue.

The treatment of congenital cystic disease of the lung depends on the character and distribution of the lesions. The importance of differentiating congenital cysts from acquired nonepithelized intrapulmonary cavities such as pneumatoceles, which may disappear spontaneously, should be borne in mind. Occasionally large fluid cysts disappear spontaneously.[2] Expansile air cysts may present a serious problem in

infancy. Due to progressive enlargement of the cyst, the normal lung is encroached upon and serious or even fatal respiratory difficulties may ensue. Attempts to deflate large balloon cysts by aspirating the contained air have been made repeatedly. The procedure is a dangerous one because in the case of the congenital cysts, particularly in infants and children, the pleural space is often not obliterated. Therefore, after the aspiration of air from the cyst, there is frequently leakage of air into the pleural space. A tension pneumothorax often results. If the patient's respiratory reserve has already been seriously encroached upon by the compression of pulmonary tissue by the cysts, the tension pneumothorax may prove fatal unless it is immediately controlled. It should not be assumed that, as the air leaks out of the hole in the cyst wall into the pleural space, the cyst will necessarily collapse. The defect in the cyst wall may also act like a check-valve mechanism and permit air to leak into the pleural space during inspiration but less readily permit air to re-enter the cyst from the pleural space during expiration. Therefore, the tension pneumothorax may merely displace the uncollapsed cyst. Moreover, aspiration may give very transient relief due to the rapidity with which the air re-accumulated in the cavity because of the check-valve bronchial communications. Occasionally closed tube drainage is satisfactory. A few large expansile cysts have been treated successfully by lobectomy or pneumonectomy in childhood. Infected cysts should be drained. Sometimes the visceral and parietal pleura are not agglutinated even over a large cyst which extends close to the chest wall, and, therefore, steps must be taken to avoid pleural contamination at the time of thoracotomy. Following drainage the cavity may or may not become obliterated. If an epithelial lining is present, this lining must be destroyed before the cavity will disappear. Many of these latter cases require removal of the cyst wall or lobectomy. In some cases the infection may destroy the cyst wall and obliteration result. Destruction of the cyst wall by caustics has occasionally been successful.



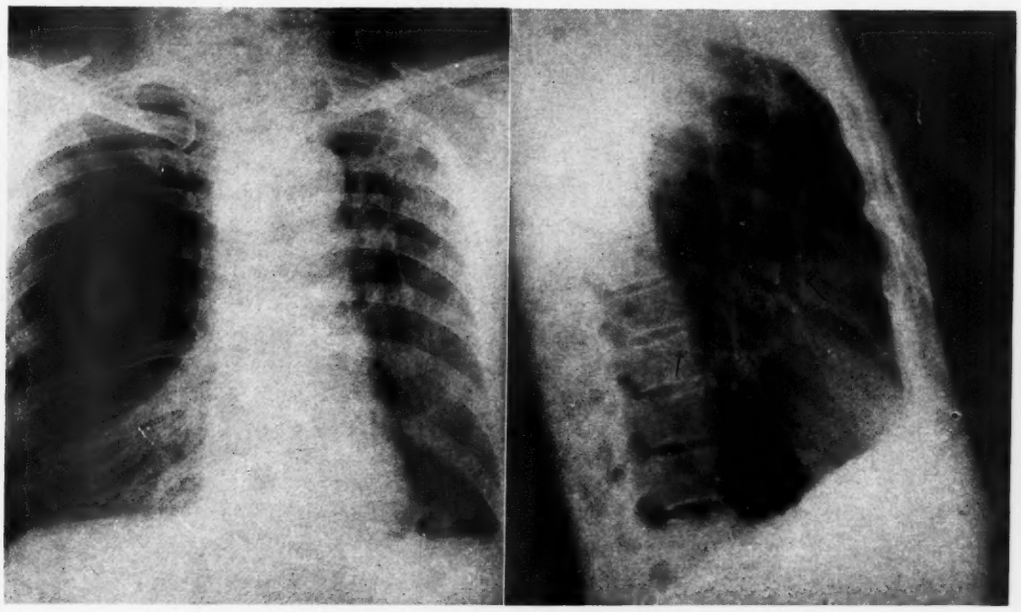
A B

Fig. 5. Bronchiectasis with bullous emphysema of right lower lobe. A, large and small bullae in costophrenic sulcus; B, bronchogram shows saccular bronchiectasis of right lower lobe. Some pneumothorax is present.

In cases of multiple cysts in one lobe or even in one entire lung, lobectomy and occasionally pneumonectomy is indicated. Large infected solitary cysts of the lung

are often erroneously diagnosed as encapsulated empyema before operation, and at times the true condition is not recognized

pleura alone in cases of chronic empyema. If the infected cyst cavity is for the most part filled with air, the roentgenogram may



A B

Fig. 6. Huge intrapulmonary air cavity in patient with chronic pulmonary tuberculosis and pulmonary fibrosis and emphysema. A, postero-anterior roentgenogram shows absence of pulmonary markings in large part of right lung field; B, outline of cyst-like space is indicated by arrows on lateral film.

during or after operation.[9] A striking characteristic of an epithelized pulmonary cyst is its failure to diminish appreciably in size over long periods of time despite adequate drainage. The roentgenologic examination is frequently of great aid in the differentiation of empyema and infected pulmonary cysts, but the two lesions cannot be distinguished by this means in some cases. The contour of the fluid pocket is often of differential diagnostic value in that the outline of a cyst is spherical or oval in both postero-anterior and lateral projections, whereas the outline of an encapsulated empyema may be triangular or fusiform and conforms more to the contour of the thoracic cage or neighboring structures in the region it occupies. Little evidence of pleural thickening may be seen on the roentgenogram of a person with a pulmonary cyst even though the infection is of many months' standing. The combined thickness of the cyst wall and pleura may be less than the thickening of the parietal

show strands of pulmonary tissue traversing the cavity, and the correct diagnosis should then be suspected. The presence of other pulmonary cysts, either fluid or air-containing, may give a clue to the correct diagnosis. It must be remembered, however, that an infected pulmonary cyst and empyema may co-exist.

At operation an absence of periosteal reaction on the inner surface of the overlying ribs may be noted in the case of an infected pulmonary cyst. This differential point is of greater significance if the infection has been present for a considerable length of time. Instead of finding the thickened parietal pleura characteristic of an empyema, the pleura may be quite thin. It may be noted that the pulmonary tissue is traversed in reaching the pus; but this finding does not necessarily mean that one is dealing with an intrapulmonary process because the approach to an encapsulated empyema may be through pulmonary tissue if an improper drainage site is

chosen, or if the empyema does not extend to the thoracic wall. Pulmonary tissue may be present or absent in the outer wall. If

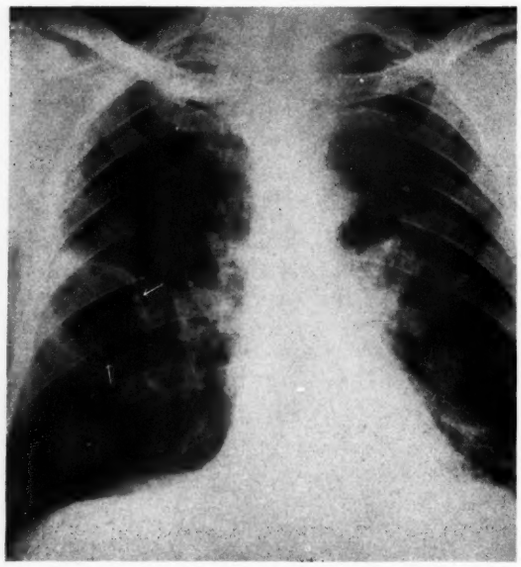


Fig. 7. Pneumatocele in adult with pneumonitis. Arrows indicate air cavity with thin wall.

present, it may be so thin that it can be recognized only microscopically. A biopsy will usually definitely establish the correct diagnosis. The wall of the bronchiogenic type of pulmonary cyst is usually lined by ciliated columnar epithelium, perhaps with areas of squamous metaplasia, and it may contain glands, smooth muscles, elastic tissue, lymph follicles and cartilage. However, infection in a pulmonary cyst may cause destruction of the epithelial lining, so that in such instances a biopsy which does not show characteristic tissue does not rule out the possible existence of a pulmonary cyst.

At the time of drainage of a pulmonary cyst it may be noted that a portion of a cavity wall has the appearance of a lattice lung with multiple bronchial fistulas. The presence of many small bronchial fistulas on the inner surface of the cavity wall should be regarded with suspicion, although many fistulas may be present in an empyema with an associated pulmonary abscess which has ruptured into the pleural space. In cysts with the appearance of a lattice lung, cord-like strands sometimes extend across the cavity. These cord-like structures resemble the septa which are seen in multiloculated pulmonary abscesses, or the adhesions which contain pulmonary tissue in cases of pulmonary tuberculosis with pneumothorax. If a biopsy of one of these strands shows it to be covered completely by respiratory type of epithelium, the intrapulmonary nature of the lesion is demonstrated. (Fig. 3.)

If the true nature of the condition is not recognized at the time of the original drainage and the case is treated as an empyema, a striking feature in the postoperative course is the failure of the cavity to diminish progressively in size despite adequate drainage for months or years and without the presence of markedly thickened pleura. An initial decrease in size usually occurs due to the fact that the contents of the cavity were under pressure before drainage was instituted. The cavity of a pulmonary cyst will maintain its spherical or oval shape after drainage in contrast to the usual elongated shape of an empyema cavity. Associated with the failure of the cavity to collapse, any mediastinal displacement to the contralateral side which was present before surgical drainage may remain to a greater or less extent. The diaphragm may not become elevated as it tends to in the healing of an empyema cavity. The smooth, shining wall of the cyst is also in contrast to the fibrotic or granulating surface of the interior of an empyema. A bronchogram following drainage of a cyst may be of aid in the differential diagnosis.

Treatment of large infected pulmonary cysts with an epithelial lining should consist first of surgical drainage of the purulent contents by thoracotomy. In cases of suspected cyst, a rib resection rather than an intercostal type of drainage should be done to enable the operator to visualize the interior of the cavity and to secure a biopsy. If the first biopsy specimen is inconclusive, another more satisfactory specimen should be obtained. With subsidence of the inflammatory process in the cyst, the smoothness of the lining wall may be-

come more apparent grossly and the biopsy examination more satisfactory.

Several reports in the literature mention unsuccessful attempts to obliterate the large cavity which persists after drainage by various forms of collapse procedures, such as thoracoplasty and phrenicectomy. Collapse procedures are definitely contraindicated as the primary method of treatment in the obliteration of these cavities; healthy lung will be sacrificed, and an incomplete collapse of the cyst will result.

The only procedures which result in obliteration of the pulmonary cavity are those which destroy or remove the epithelial lining. Chemical or electrosurgical cauterization of the epithelium are rarely effective. Furthermore, this method does not seem practical if numerous free bronchial communications are present and if there is a well developed epithelized wall with muscle and elastic tissue.

Removal of the cyst wall is the most certain method of treating the epithelized pulmonary cyst. This may be done by removal of the lobe containing the cyst or by enucleation of the cyst itself. If no definite line of cleavage around the cyst is present, lobectomy is the preferable procedure; otherwise, serious bleeding or air embolism may result. Lobectomy may be technically more difficult than the usual lobectomy for bronchiectasis. Anomalous vessels of large size, especially branches penetrating the diaphragm, may be encountered. The patients are usually children or young adults, and they are, therefore, at an age period when major intrathoracic procedures are borne well. With the infected contents of the cyst drained, ample time should be taken to permit subsidence of the inflammatory reaction and to improve the patient's general condition before radical surgery is employed.

CYSTIC BRONCHIECTASIS

Cystic bronchiectasis is included under pulmonary cysts by many authors. Some classify bronchiectasis as one form of congenital cystic disease of the lung. Yet, we definitely know that cystic bronchiectasis may also be an acquired lesion secondary to bronchial obstruction by tumor or other process. It would seem best to classify cystic bronchiectasis as a form of bronchiectasis rather than consider the lesion as a type of pulmonary cyst.

Cystic bronchiectasis is treated similarly to other types of bronchiectasis. Lobectomy or pneumonectomy is indicated if the process is not too wide spread to warrant surgical excision of the involved areas. If untreated, the prognosis is not good because the extensive structural changes in the lung leave a fertile field for secondary infection. In some cases, however, the symptoms may be relatively slight for long periods of time.

ACQUIRED INTRAPULMONARY CAVITIES ORIGINALLY PRODUCED BY INFECTION

Cavitation in the lung parenchyma may remain indefinitely in cases of pulmonary suppuration even after the infection has subsided. The long persistence of the cavity of a pulmonary abscess after subsidence of the inflammatory process is usually due to the fibrosis of the adjacent lung, but is occasionally caused by the ingrowth of epithelium into the cavity from the communicating bronchi. The cavity may be lined by cuboidal, columnar or metaplastic squamous epithelium. The epithelization of the cavity may be partial or complete. A similar process may sometimes occur in an old tuberculous cavity in which the active tuberculous involvement has disappeared. Histological examination of a portion of the cavity wall is necessary to demonstrate whether epithelization of the cavity has occurred. A nonepithelized cavity remaining after pulmonary suppuration may have similar clinical findings.

If the patient has been studied radiographically during the course of the acute pulmonary suppuration, the origin of the intrapulmonary cavity may be readily evident. In such cases the typical roentgen findings of a pulmonary abscess cavity

with a fluid level and surrounding pulmonary infiltration may have been observed. With improvement of the infection the cavity may become empty and the surrounding infiltration clear. Whereas in most instances the cavity progressively diminishes in size as the infection subsides, in occasional cases the cavity persists. This is most likely to occur in cases of chronic lung abscess or in those cases in which there is considerable fibrosis of the adjacent pulmonary tissue which interferes with expansion of the surrounding lung. Epithelium may extend from the communicating bronchi into this persisting cavity. The epithelization then in turn prevents cavity obliteration. This process may occur both in pulmonary abscesses which have subsided through bronchial drainage or following surgical drainage of the purulent contents.

If the patient is first observed after the cavity has become empty of pus and the surrounding pulmonary infiltration has disappeared, it may be difficult to determine the mode of development of the cavity. It is under such circumstances that confusion with congenital cysts may arise. It may be erroneously assumed that the infection was superimposed on a preexisting congenital pulmonary cyst. (Fig. 4.) At times it may be difficult or impossible to differentiate roentgenographically an epithelized cavity from a pneumatocele. The former, however, usually has a thicker wall and does not change much in size, whereas the latter has a thin wall and may fluctuate considerably in size or disappear.

The epithelized cavity *per se* requires no treatment. At any time, however, the cavity may become re-infected through its bronchial communication. The pulmonary tissue surrounding such cavities, moreover, is usually chronically diseased. Pulmonary fibrosis, emphysema and bronchiectasis are frequently present. For these reasons, lobectomy or pneumonectomy is often indicated. Drainage of the cavity alone will not result in healing unless the secondary infection destroys the epithelial lining. It is rarely feasible to excise the cavity wall or destroy the epithelium by cautery or caustics.

EMPHYSEMATOUS BULLAE

In pulmonary emphysema there is fragmentation of the elastic tissue with rupture of the interalveolar septa. In most cases of pulmonary emphysema the number of alveolar septa which rupture in a given area is sufficient to produce only a small cavity. The disruption of the interalveolar septa may be fairly uniform throughout the lung although there is a tendency for the process to be pronounced in certain areas, especially along the border of the lung and in the pulmonary apices. In cases of diffuse emphysema, the roentgenogram shows increased radiolucency throughout the pulmonary field. If, on the other hand, extensive disruption of the interalveolar septa occurs in a localized portion of the lung, a large cavity, called a bulla, is produced. (Fig. 5.)

Obstruction to the egress of air from an emphysematous area in the lung probably plays a rôle in the development of an emphysematous bulla as it does in the formation of a pneumatocele. In the case of the emphysematous bulla this increased intracavitary pressure is transient as during coughing. If the cavity constantly had a positive intracavitary pressure, we would classify the lesion as a pneumatocele even though it occurred in an emphysematous lung. The reason for the distinction is that therapeutic measures of a deflating type would be of value only if positive intracavity pressure existed.

One or more such bullae may be present. The cavity is lined by the walls of the surrounding alveoli and there is no true epithelial lining. The bullous cavity has a rather poor communication with the bronchial tree by means of one or more small bronchi. The bulla shows on the roentgenogram as an area of even greater radiolucency than the surrounding emphysematous lung. If the bulla is large, there may be an absence of pulmonary markings

in that region. In some instances the outline of the bulla may be clearly defined on the roentgenogram, but frequently the borders are indistinct, at least in certain projections. (Fig. 6.) Emphysematous bullae are, therefore, an advanced form of emphysema, and it seems more appropriate to employ the term emphysematous bulla rather than call a large bulla a pulmonary cyst. Emphysematous bullae have to be differentiated from localized pneumothorax, pulmonary blebs and pneumatoceles. The differentiation of an emphysematous bulla and a localized pneumothorax is frequently difficult. The history and physical findings are usually of little aid. The roentgenogram is of greatest help. The outline of the bulla is round or oval and it may not extend to the chest wall as a pneumothorax does. In certain projections, especially the lateral view, the outline of the air cavity may be clearly defined as a thin line. Although it is impossible to draw a definite line of distinction between emphysematous bullae and pneumatoceles, attempts should be made to differentiate the two conditions. If the intrapulmonary air cavity is produced chiefly or entirely by the disruption of interalveolar septa and inflation of the cavity by a check-valve mechanism in the bronchus is absent or plays a minor rôle, the lesion is classified as an emphysematous bulla. If, on the other hand, the size of the cavity is largely due to hyperinflation of an originally small cavity produced by disruption of a few interalveolar septa or a small area of pulmonary destruction, the lesion is classified as a pneumatocele. The differentiation of bullae and pneumatoceles is of practical importance in a consideration of the clinical course of treatment. As a result of the different mechanics of the two conditions, emphysematous bullae do not disappear but tend to increase slowly in size, while pneumatoceles may rapidly increase or diminish in size and not infrequently disappear completely and spontaneously.

Kaltreider and Fray[7] studied the pathological physiology of the lungs in six patients with air-filled pulmonary cavities. The patients had pulmonary fibrosis and emphysema with emphysematous bullae of varying size. In half of the cases the total lung capacity, as well as the vital capacity was reduced while the residual air was only slightly increased. These findings indicate that in these cases the volume of the cyst is not included to a significant degree in the residual air. Therefore, the emphysematous bullae were poorly ventilated because of poor communication with the bronchial tree. In the remaining cases there was a marked decrease in vital capacity and a corresponding increase in the residual air with a relatively normal total capacity. The findings in these latter cases are typical of those of obstructive pulmonary emphysema.

The treatment of bullous emphysema is unsatisfactory. As it is a part of pulmonary emphysema and fibrosis, the involvement of the lungs is usually widespread. The patient may have dyspnea and even cyanosis. Sometimes there is marked respiratory insufficiency. Secondary to the pulmonary fibrosis there is hypertension of the pulmonary circuit and frequently right ventricular hypertrophy. Needling of emphysematous bullae is dangerous because a tear in the thin wall of the bulla may cause a tension pneumothorax.

Although emphysematous bullae have been discussed in this paper on pulmonary cysts we have done so chiefly in order to point out the difference between a bulla and other lesions which may have been classified as pulmonary cysts. It seems best to consider an emphysematous bulla as a locally advanced stage of pulmonary emphysema rather than call the bulla a cyst after it attains a certain size.

PULMONARY BLEBS

Pulmonary blebs are collections of air in the interstitial tissues of the lung. The air is localized in the interlobular connective tissue and just beneath the pleura. Pulmonary blebs are caused by the rupture of alveoli and the dissection of air

along the tissue planes of the lung. The pulmonary bleb may rupture and cause a pneumothorax. There may be associated mediastinal emphysema due to the dissection of air through the pulmonary hilum into the mediastinum. The process is acute and usually can be easily differentiated from other types of cyst-like spaces in the lung.

PNEUMATOCELES

A pneumatocele is a hyperinflated intrapulmonary cavity produced by the marked distention of a defect in the pulmonary parenchyma. The pneumatocele follows an interstitial pneumonitis with an inflammatory involvement of a small branch bronchus. As a result of the inflammatory narrowing of the bronchus a check-valve mechanism is produced and the pulmonary segment which communicates with the obstructed bronchus or bronchiole is tremendously dilated. (Fig. 7.) There is some disruption and destruction of the interalveolar septa both as a result of the increased intra-alveolar pressure and as a result of the interstitial inflammation. The degree of rupture of interalveolar septa is probably influenced by the elastic property of the lung. If the inflammatory process has occurred in the lung of a child and the elastic qualities of the lung were normal before the infection, there would be less fragmentation of interalveolar septa. If the pneumatocele occurs in a lung which has already lost some of its elasticity due to emphysema, rupture of interalveolar septa would probably play a greater rôle in the production of the pneumatocele. In support of this contention it may be pointed out that pneumatoceles may disappear more quickly in infants and children than in adults.

There is no definite line of distinction between a pulmonary abscess cavity and a pneumatocele. In a pulmonary abscess the cavity produced is chiefly caused by the necrosis and liquifaction of a somewhat comparable amount of pulmonary tissue. The rounded contour of the cavity is partly determined by the elastic qualities of the adjacent pulmonary tissue. In some cases of pulmonary suppuration in which cavitation is present, there is no close correlation between the size of the pulmonary cavities seen on the roentgenograms and the amount of pulmonary tissue which has undergone necrosis. A small abscess cavity may become inflated by a check-valve mechanism of the bronchus into a large cavity. Therefore, a small area of pulmonary necrosis may produce a small cavity which is then inflated to a large size. It may be difficult to judge how much pulmonary destruction has occurred. This type of process seems to be considerably more common in cases of aerobic pulmonary infection than in anaerobic infections. In the latter group there is likely to be more rapid and extensive pulmonary destruction. Inflation of small areas of pulmonary excavation into large cavities is probably seen in a higher percentage of cases of pulmonary infection in infants and children than in adults.

These cases frequently run a somewhat characteristic course. The child has an acute respiratory infection with cough and fever. Roentgenograms show areas of consolidation. The cough may or may not be productive. On serial roentgenograms one ro more cavities of varying size may appear in the area of pulmonary infiltration. A large cavity may appear within a few days without the expectoration of any significant amount of sputum. In other words there is no clinical evidence to suggest the necrosis and liquifaction of considerable pulmonary tissue in a few days. The cavity may be entirely air-containing or a fluid level may be present due to the presence of both air and fluid in the cavity. Usually the fluid level is rather low; the presence of a high fluid level in the cavity would speak more in favor of pulmonary suppuration playing a more prominent rôle than hyperinflation. Frequently the finding of the cavitation is a surprise to the clinician because not infrequently the clinical signs of pulmonary infection may already be

slight at this time. Another roentgenogram taken a few days later may show marked enlargement in the size of the cavity. By this time the patient may be afebrile and almost free of pulmonary symptoms. If there is some cough present, it is usually productive of little sputum. In other words the clinical picture is rather inconsistent with a large acute pulmonary abscess. At this time the radiologist reports the presence of a large abscess cavity. In view of the rapid increase in size of the cavity as seen on the roentgenogram it may be concluded that rapid destruction of pulmonary tissue is taking place and surgical drainage may be considered advisable. The clinical significance of a large intrapulmonary cavity produced by tissue necrosis is very different from a large cavity which owes its size to hyperinflation. Pneumatoceles do not require surgical drainage. The correlation of the clinical course with the serial roentgenographic findings will usually permit a proper differentiation.

In infants and children especially, pneumatoceles may attain huge size. One or more pneumatoceles may occupy almost an entire hemithorax and markedly compress the surrounding pulmonary tissue. Peirce and Dirkse[10] have followed the course of such pneumatoceles over a period of time. Some pneumatoceles may disappear in a few weeks while others persist and often fluctuate in size over a period of months or years, although the patient is asymptomatic. The pneumatocele may disappear spontaneously after it has been present for months or even years. At any time while the pneumatocele is present, however, a flare-up of the pulmonary infection may occur.

A pneumatocele usually requires no surgical treatment in contrast to congenital pulmonary cysts and huge abscess cavities from which it must be differentiated. Extirpation of a lobe or the entire lung has been performed in a few cases of pneumatocele, but the advisability of lobectomy or pneumonectomy seems questionable if the lesion is truly a pneumatocele. It is only if persistent marked compression of adjacent pulmonary tissue causes respiratory difficulty that surgical therapy is warranted. Then decompression of the pneumatocele, performed in such a way as to avoid a tension pneumothorax, is indicated. Intracavity suction by catheter may be satisfactory.

Caffey[3] has stressed the importance of differentiation of the cavitation of a pulmonary abscess from the regional obstructive pulmonary emphysema associated with infections of the respiratory tract in infants and children. After partial contraction the cavities may re-expand without the recurrence of inflammatory changes in the adjacent lung and without re-accumulation of fluid in the cavities or signs of infection. If the size of the cavity were regarded as a criterion of pulmonary destruction, it would be erroneously decided that the pulmonary suppuration was progressing and surgical measures were indicated. A similar situation is occasionally encountered in the aerobic pulmonary infections in adults.

PARASITIC CYSTS

Parasitic cysts of the lung are uncommon in North America.[11] Echinococcic or hydatid cysts are the most important type of pulmonary cyst. Hydatid disease is most common in the Mediterranian region, Australia, New Zealand and parts of South America.[4] Of the small number of cases of echinococcic cysts of the lung which have been reported from the North American continent, in all except a few instances the disease was probably acquired while living in another part of the world. Hydatid cysts usually involve the liver, but the lungs are the second most frequent site of involvement. Hydatid cysts of the lung may be primary or secondary; the primary cyst is the more common. The right lung is involved more frequently than the left. The base of the right lung is a frequent site of hyatid cysts due to extension of the disease from the liver through the diaphragm.

An intrapulmonary echinococcic cyst may cause no symptoms for a long period of time unless rupture into the bronchus or the pleural cavity has occurred. In some cases cough is an annoying symptom. Hemoptysis is common. Pain may be present and dyspnea may be produced by a large cyst. The intrapulmonary hydatid appears on the roentgenograms as a round or oval area of increased density. The borders of the shadow are less sharply defined than in the case of solid tumors. The shadows may be single or multiple. There may be evidence of associated pleural involvement. As the pulmonary hydatid enlarges, it may rupture either into a bronchus or the pleural cavity. If rupture into a bronchus has occurred, a fluid level may be present within the cyst. When an echinococcic cyst ruptures into the bronchial tree, salty-tasting, clear fluid may rush into the mouth, and portions of cyst wall may be coughed up. Godfrey[6] states that deeply placed peribronchial cysts frequently undergo spontaneous cure if they rupture into a bronchus. After a hydatid cyst has ruptured into a bronchus, the cyst may become secondarily infected and the sputum foul. Then the patient presents the picture of pulmonary suppuration. Echinococcic cysts may be differentiated from nonparasitic fluid containing cysts, intrapulmonary tumors or abscesses, localized empyema and mediastinal cysts. The precipitin test, the intradermal test, and the complement fixation test combined with the demonstration of an eosinophilia, which is present in half the cases, may aid considerably in the diagnosis. Aspiration of the hydatid cyst is contraindicated because of the danger of spilling the contents of the cyst into the pleura and because of the possibility of anaphylaxis. If the cyst has ruptured into a bronchus, hooklets may be demonstrated in the expectorated material.

Most authors recommend surgical therapy for the large echinococcic cysts. Early surgical treatment is also indicated in small, subpleural echinococcic cysts because there is considerable risk of spontaneous rupture into the pleural cavity and the chance of spontaneous cure by rupture into a bronchus is poor. Surgical intervention is not usually recommended for the small deeply situated cyst near the pulmonary hilum because of the frequency of spontaneous cure in this group following rupture into a bronchus. In the removal of an echinococcic cyst it is important to avoid contamination of the pleural space. Some surgeons recommend complete extirpation of the cyst with closure of the chest without drainage. A one-stage operation may be performed with suture of the pleural layers if adhesions are not present. In the two-stage operation the first stage is given over to the creation of adhesions between the lung and the pleura, and the cyst is removed at the second operation.

CONCLUSIONS

The indiscriminate grouping of a variety of pulmonary lesions as pulmonary cysts leads to confusion. The various lesions should be classified according to etiology and pathology as far as this is possible.

Many acquired lesions have been incorrectly assumed to be congenital; radiographic differentiation of congenital and acquired cysts is often not possible.

Unless the mechanism of development and the pathological characteristics of a pulmonary cavity are considered, the correct therapy may not be employed.

Cavities produced by pulmonary destruction must be differentiated from cavities produced chiefly by hyperinflation. The treatment of epithelized and non-epithelized cavities frequently differ.

Surgical therapy plays an important rôle in the management of certain types of pulmonary cysts.

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A REALLY simple goiter, . . . without either toxicity or degeneration, is really only a deformity caused by the distention of follicles with colloid, a physiologic disturbance capable of being influenced by therapeutic measures.

PRINCIPLES IN THE MANAGEMENT OF PULMONARY ABSCESS*

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EXCLUDING neoplasm, pulmonary abscess is one of the most serious diseases of the lung. The mortality in reported series ranges from 30 to 50 per cent. Sweet[1] reported a mortality rate of 34 per cent in 124 cases; Cutler and Gross[2] reported 38 per cent in eighty-five cases; Brunn[3] reported 60 per cent in 205 cases. Many other patients who did not actually die in the hospital were left as chronic pulmonary invalids due to irreparable damage to the lung by the infectious process. It is rare to find a reported cure rate of 50 per cent or over. In the past few years, Neuhof and associates,[4,5,6] and Overholt and Rumel[7] have shown markedly improved results in acute cases treated by prompt surgical drainage. Neuhof[6] and his group had four deaths in 104 acute abscesses, while Overholt and Rumel[7] reported two deaths in thirty-five "simple" (mostly acute) abscesses. Even more important was a cure rate of 94 per cent in Overholt and Rumel's series.

It is the purpose of this paper to add evidence to that contained in the recent reports, to indicate a broadening of the indications for surgical treatment, and to support the view that pulmonary abscess should be considered primarily a surgical disease from its onset. Conclusions are based upon twenty-five consecutive cases treated by the author with but one operative death.

DEFINITION

Pulmonary abscesses due to the tubercle bacillus or secondary to neoplasms are not included in this study. Likewise, multiple lesions on a definitely bronchiectatic basis or as a part of a generalized pyemia are excluded. Certain of the rare conditions such as infections due to Friedlander's bacillus or to fungi are not within the scope of this discussion. It does include, however, all other types and no effort has been made to consider separately various classifications such as acute or chronic, simple or complicated, putrid or aputrid, localized or diffuse, single or multilocular, or pulmonary gangrene. The underlying pathological process is the same in each instance; and although the manifestations may differ, the principle of early adequate drainage has been followed in all. However, the fulminating diffuse lesions or the frankly gangrenous processes will not yield as good results as are obtained in the treatment of early, acute, single abscesses.

BACTERIOLOGY

The bacterial flora of pulmonary abscess is variable. Usually a mixture of staphylococci and streptococci is found with frequent other associates. Both aerobic and anaerobic organisms are present in most instances. Specific therapy either with neoarsphenamine or one of the sulfonamide drugs has been disappointing, and bacteriology for the present has been relegated to a place of minor importance. Details of treatment may have to be varied according to the etiological organism but the principles of treatment are the same for all types.

It appears that most of the destructive and necrotizing organisms are either strict or facultative anaerobes. Reports on the bacteriology, unless including careful anaerobic cultures, are not significant. Many abscesses will be found to contain sterile pus if only aerobic cultures are taken. Although some of the organisms are very

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virulent under certain conditions, many are easily destroyed by an unfavorable environment. Studies of these organisms must take such factors into account. It is our practice to aspirate secretions from the abscess into a test tube at operation. This material is then transferred immediately to a thermos bottle and kept at body temperature until it reaches the laboratory and is plated on various media. Such cultures most always show a mixture of organisms with staphylococci and streptococci predominating. Vincent's organisms may be recovered from the gums of a patient with a lung abscess, but they are not frequently encountered in the abscess cavity itself.

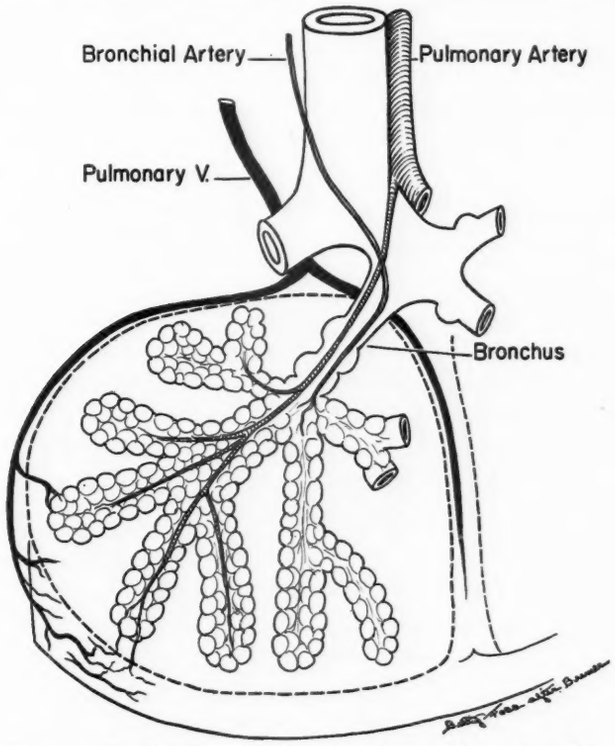

Fig. 1. A diagramatic drawing illustrating the close proximity of the pulmonary and bronchial arteries to the corresponding bronchus.

PATHOLOGY

We subscribe to the belief that aspiration of infected material into a bronchus is the primary cause of most pulmonary abscesses.[8,9,10] This material may be inhaled from infected gums during sleep or at operation. It may be detritus at the time of tonsillectomy or tooth extraction. Some abscesses follow pneumonia, especially if the pneumonic process is due to a staphylococcus. Although other factors, such as a septic embolus, are occasionally responsible, it seems likely that all but the exceptional case are due to aspiration.

When the bronchial lumen to a segment of lung becomes occluded, the portion of lung beyond becomes atelectatic and a perfect environment for anaerobic organisms is established. The spread of infection to the peribronchial structures impairs the blood supply to the segment beyond and hence in the course of a few days the area may become necrotic and symptoms of lung abscess appear. The close relationship of the bronchus and blood supply to a pulmonary lobule is shown diagrammatically in Figure 1. Peribronchial infection need not be extensive to include the accompanying branch of the pulmonary artery.

Mathes, Holman and Reichert[11] found that bronchial occlusion in the dog was universally fatal unless the foreign body

(sponge) was removed. Likewise pulmonary embolus although well tolerated in the absence of infection was always followed by abscess formation in the presence of infection. A septic bronchial obstruction by extension of the infection to the accompanying pulmonary artery resulting in thrombus formation can provide this serious combination.

Infection in a pulmonary segment calls forth the usual protective inflammatory wall. This protective wall is seen on inspecting the interior of the abscess cavity at operation and is further evidenced by the almost universal finding of pleural symphysis over the peripheral portion of the abscess. When this protective process is not sufficient, extension of the necrotizing pulmonary process to the surface of the lobe results in empyema.

Pulmonary abscess is a segmental lesion as shown by Glass.[12] The causative factor is located centrally and is the source of the diminished blood supply to the peripheral portion of the segment. Pulmonary arteries are end arteries, thus the periphery of a lobule has the poorest blood supply. This region, therefore, is the first to undergo dissolution and the abscess first becomes manifest in this area. The periphery of the lobule forms the periphery of the lobe, thus abscesses due to bronchial occlusion must be in contact with either the chest wall, interlobar fissure, diaphragm or mediastinum. Abscesses which have been referred to as "central" are those not facing the lateral chest wall, or are so located as to project "centrally" on the postero-anterior roentgenogram.

BASIS FOR EARLY SURGICAL DRAINAGE

A certain percentage of lung abscesses will regress spontaneously and many of these may give no further symptoms after a period of medical management. The exact number that will do so is undeterminable but it is probably in the vicinity of 20 to 35 per cent. Medical management includes a good hygienic régime with postural drainage, medicaments, bronchoscopic aspiration and pneumothorax. The latter, however, has been almost abandoned due to the high incidence of serious or even fatal complications. Reported series include King and Lord's report;[13] 40 per cent of the patients were cured and the mortality was 35 per cent among those treated medically. Rosenblatt[14] reports 7 per cent cured and 47 per cent dead. Jackson and Judd[8] report 42 per cent cured and 29 per cent dead by medical measures including bronchoscopy. However, in the series of Jackson and Judd there were thirty-seven other patients who were referred for surgical treatment, eighteen or 48 per cent of whom died, making a true mortality rate of 36 per cent for the whole series.

When medical management should cease in a given case and surgical treatment be instituted is a difficult question to decide. Most authors favor a period of six weeks to three months before considering surgical drainage. The results of surgery in such a group are likely to be poor, as those patients have suffered the effects of a long septic illness. Cutler and Gross[2] reported a mortality of 38 per cent in their group of eighty-five surgical cases, and Allen and Blackman[15] reported a mortality of 30 per cent in ninety-four cases. These figures based as they are on a group of poor risk patients after protracted medical management do not give a true picture of the possible surgical results. Assuming that of one hundred cases of lung abscess, thirty will recover spontaneously and 60 per cent of the remaining seventy will get well following surgery, it still leaves a 28 per cent mortality for the group as a whole. No one can predict which case will heal spontaneously or when a given case will become complicated. If surgery can cure more than 72 per cent of the group as a whole by operating upon all patients early in the disease, it would be better to operate upon all as in the end more patients would be living and well.

The same line of reasoning applies to the time at which operation should be carried out. If one cannot foretell with

certainty the stage at which complications will develop, all patients should be operated upon before any enter the complicated state. Rives, Major and Romano[16] in a study of one hundred fatal cases reported that 75 per cent of that group were frankly hopeless within one month of the time of onset. Sweet[1] reported four deaths out of eleven patients operated upon who had entered the hospital within four weeks of onset. He further states that "of 14 cases in which death occurred without operation, 10 died in less than 2 months after the onset of the disease." Jackson and Judd[8] found that eleven of twenty-five deaths occurring on "conservative" therapy were within two months of onset.

The principles of drainage of pulmonary abscess are essentially those which apply to an abscess in any other location. These are thorough and adequate drainage of the process at the earliest time and with as little disturbance of surrounding normal structures as possible. The small and inconstant lumen of an inflamed bronchus does not provide adequate drainage for a pulmonary abscess. A small opening of a comparative size would be considered wholly inadequate for an abscess located elsewhere. The frequent finding of a large amount of thick inspissated material within the abscess at the time of operation is further proof of the inadequacy of bronchial drainage. How can open drainage be provided with safety? It is necessary to take advantage of the superficial location of the lesion and the adhesions formed by the process. This can be done if there is accurate roentgenological localization.

ROENTGENOLOGICAL LOCALIZATION

Accurate preoperative roentgenographic localization is vitally important. Pleural symphysis is most secure and extensive over that portion of the abscess that is nearest the periphery. This site must be determined roentgenologically.

Preliminary fluoroscopic study will aid in determining the most advantageous position for taking the roentgenographic plates. Postero-anterior, lateral and anterior oblique views will usually be sufficient. It may be necessary to supplement these conventional views by overexposed or Bucky films, or projections of varying obliquity.

After the location of the abscess has been determined, its exact relationship to the chest wall should be checked by a repeated series of films after placing an appropriate radio-opaque marker. In placing this marker allowance for the shift of the chest wall structures must be made. The marker should be placed with the patient in the same position he will assume upon the operating table. Neuhof's practice of injecting a mixture composed of 2 to 3 m. of lipiodal and a dye into the intercostal muscles is helpful. The lipiodal will be seen on the films and the dye provides a visible landmark at operation. We have found that lamp black is more suitable than some of the dyes in that it remains visible in the tissues for a longer time.

ONE-STAGE OPERATION PREFERABLE

The possibility of contamination of the pleural space or a dessicating infection of chest wall structures are advanced as reasons for employing a two-stage procedure. It has been found by Neuhof,[4,5] Overholt and Rumel,[7] and confirmed by our own observations that if the abscess faces the thoracic cage, pleural symphysis will be found if the lesion is properly localized. The area of adherence may not be large but will be sufficient for safe drainage. It is interesting to note that in the report of Overholt and Rumel[7] empyema occurred as a postoperative complication four times as often after the two-stage drainage as after a one-stage procedure. By sealing off the fascial planes as described below, the fear of a dissecting cellulitus is more theoretical than practical. By obviating the former objections to the one-stage procedure this method becomes applicable to all abscesses except those facing an interlobar fissure, the diaphragm or medi-

astinum when chest wall adhesions may not be present.

The lapse of time between stages of a two-stage procedure may be hazardous. Even shortening the usual interval to only one or two days does not remove the risk. Occasionally, the first stage operation is followed by an exacerbation of the process or an extensive spread, thus converting a simple lesion to a complicated problem.

ANESTHESIA

It is our practice to employ procaine hydrochloride paravertebral block plus local infiltration.

Caution must be exercised to prevent spill-over of purulent material to other parts of the same lung or to the contralateral lung during operation. It is usually necessary to have the patient lie with the affected lung uppermost thus favoring "crossfire" spread. Such a complication can be minimized by use of a rather steep Trendelenburg position and close observation by the anesthetist, who should encourage the patient to cough and raise any material that may drain into the tracheobronchial tree. The use of positive pressure oxygen administered by means of a properly fitted face mask provides a further safeguard. This is begun as soon as the chest wall drainage path has been prepared. Maintenance of positive intrabronchial pressure causes air to flow from the interior of the tracheobronchial tree toward the exterior through the bronchial fistula communicating with the drainage tract. This blows any discharge from the abscess outward rather than allowing it to drain internally. Maintenance of the positive intrabronchial pressure furthermore tends to keep the periphery of the lung and the chest wall in apposition and lessens the likelihood of tearing friable pleural adhesions. The positive pressure is maintained until the abscess is drained, packed, the dressing applied, and the patient placed upon his operated side.

OPERATIVE TECHNIC

The patient is placed on the table so that the site for the incision is conveniently located. Marked Trendelenburg position is used to favor drainage of bronchial secretions and to minimize the possibility of cerebral air embolism. The site of incision depends entirely on the location of the most peripheral portion of the abscess. It is placed so as to expose the rib directly over the center of the lesion. A five-inch skin incision is usually sufficient. If the operative field is in the axilla, a Y-shaped incision may be used to advantage as this prevents too rapid closure of the chest wall structures. The skin and subcutaneous tissues and muscles are divided down to the underlying ribs. A four-inch segment of the rib is subperiosteally resected, the ends sealed with bone wax and the corresponding intercostal bundle is ligated at either end. The intercostal nerve accompanying the rib above is crushed. With a continuous suture of fine chromic catgut, the subcutaneous fascia and intercostal structures are approximated so as to seal off all fascial planes. (Fig. 2.) At this point the anesthetist begins the administration of oxygen under moderate positive pressure so as to keep the lung and chest wall in apposition. An incision is made through the periosteal bed until the lung is encountered. This establishes by actual inspection the presence or absence of pleural adhesions. If they are found, one may proceed with drainage. If not, localization has probably been inaccurate or the lesion does not point on the chest wall. It is wise then to place a piece of gauze with a metal marker attached, such as a Michel skin clip, in the incision, pack the wound and re-examine the patient roentgenographically to check the localization. If adhesions are present, a 17 or 18 gauge needle attached to a syringe is introduced 1 to 2 cm. into the underlying lung. If the localization is correct, pus or foul-smelling air should be obtained immediately. The needle is left in place as a guide and with the actual cautery a

moderate-sized opening is made in the wall of the abscess. (Fig. 3.) Large openings are to be avoided as they may extend beyond

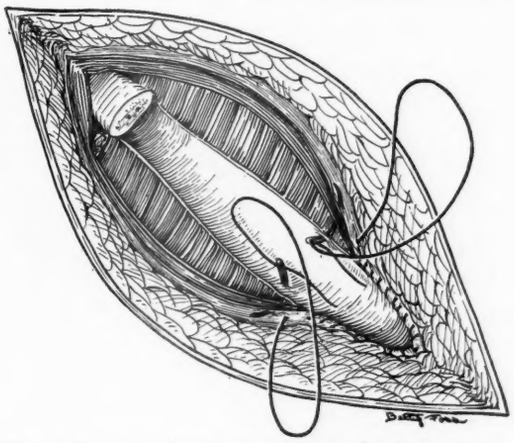

Fig. 2. Drawing to show suturing of subcutaneous fascia to the periosteal bed. A segment of rib has been subperiosteally resected. The accompanying intercostal vein, artery and nerve have been ligated and divided.

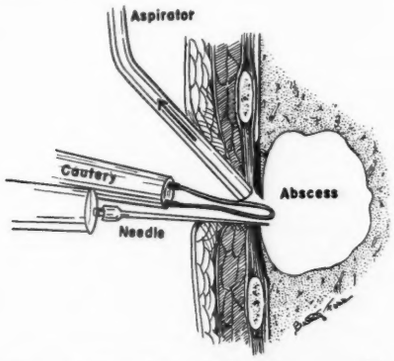

Fig. 3. Opening of the abscess with the cautery after aspiration with a needle. Note the sucker which removes smoke from the operative field and prevents spillage of the contents of the cavity.

the zone of pleural adhesions. A suction tube removes smoke from the operative field, and prevents gross contamination of the wound by the contents of the cavity. The cavity is evacuated with the sucker and material obtained for culture. With as little manipulation as possible, the cavity is cleaned out and packed lightly with dry gauze. It is our practice to place 4 to 5 Gm. of powdered sulfathiazole into the abscess, and another 1 to 2 Gm. are sprinkled into the wound to discourage bacterial growth.

A piece of rubber dam with an opening to allow drainage is sewn to the edge of the drainage tract to make subsequent dressings easy to change. (Fig. 4.) This prevents the gauze pack from becoming adherent to the skin and subcutaneous structures. A dressing is applied and the patient turned upon the operated side, and then and only

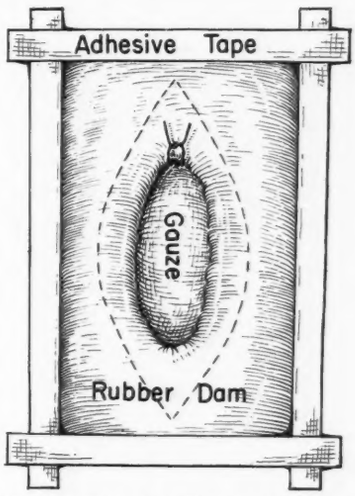

Fig. 4. Drawing illustrating the use of rubber dam to prevent adherence of the gauze pack to the wound. The rubber dam is sewn down to the intercostal structures.

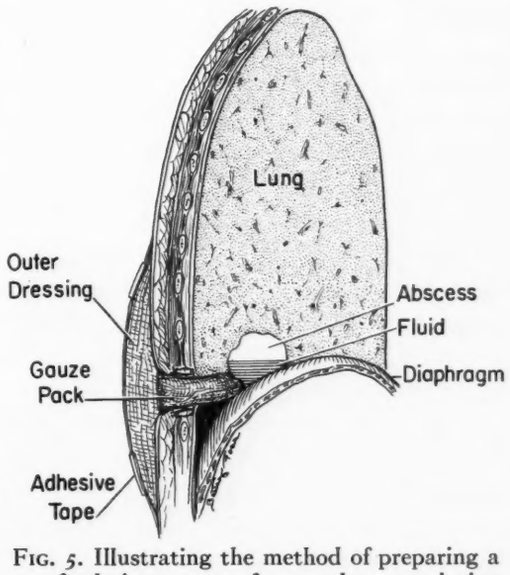

Fig. 5. Illustrating the method of preparing a safe drainage tract for an abscess pointing on the diaphragm. A segment of rib has been removed. The gauze pack inserted to the border of the abscess will cause adhesions to form, thus sealing off the pleural space.

then is the positive pressure oxygen discontinued.

TECHNIC FOR ABSCESSES NOT FACING THE THORACIC WALL

Abscesses facing the interlobar fissure, diaphragm or mediastinum may demand a two-stage drainage in order to provide a safe drainage tract through a nonadherent pleural space. The site of drainage is selected that is nearest to the underlying abscess. The operation is carried out as in the one-stage procedure to and including the incision of the pleura. The costal pleura may be found nonadherent. The space between the chest wall and the abscess is packed with a strip of gauze as illustrated in Figure 5. Two to five days later, the pack is removed and the abscess opened and treated as in the one-stage operation.

MULTIPLE ABSCESSES

Many abscesses become multiloculated. If the loculations all originated as direct extensions from the original process, they will be found to communicate with the main cavity and thus all may be drained at one time. In some cases adequate drainage may not be obtained and subsequent cauterization becomes necessary. This usually can be carried out through the original approach. Pulmonary suppuration following staphylococcic pneumonia may present a complicated problem. One or more segments of the lobe usually contain multiple abscesses filled with thick, almost gelatinous pus which drain poorly through the bronchi. The entire segment may have to be destroyed by repeated cauterizations. This may necessitate an approach from more than one avenue.

Distinct multiple abscesses in separate parts of the same lobe or in different lobes will be found in some cases when first observed. (See Cases 11, 12, 16, 19 and 21.) These must be drained through separate approaches at separate operations, attacking the largest or most acute process first.

In special instances in which the process is of long duration with marked pulmonary fibrosis and accompanied by extensive bronchiestasis, resection of the involved lobe or lung may be indicated. In general, the results of lung resection for acute abscesses are encouraging but still entail a quite high mortality.[1,7] Preliminary drainage to enable the patient to improve may be a necessary first step.[1]

POSTOPERATIVE MANAGEMENT

Care of the operative site is most important. In general, the original dressing is left in place from four to seven days. Plain dry gauze seems to be the best packing material. If the secretions are malodorous, the responsible anaerobic organisms can be quickly brought under control by packing the cavity daily for a few days with gauze soaked in zinc peroxide. The frequency of change of the pack is determined by the amount and character of the discharge. Dusting the inside of the abscess with sulfathiazole has been found effective in combatting bacterial growth. As the healing process progresses, the walls of the cavity close in and when it becomes too small to pack easily, a soft rubber tube is inserted and fastened securely in place with adhesive strips to a safety pin which is passed through the end of the tube. The caliber of the tube can be diminished as the sinus decreases in diameter. Withdrawal of the tube is not to be hurried. It may take several months for the healing process to be completed. The tube should not be less than 1 or 2 cm. shorter than the sinus. It must be left in place until the bronchial fistula has closed. This will take place spontaneously in all but a very few cases. After waiting several months, it may be necessary to resort to a muscle plastic closure of the bronchial fistula. This rarely has been found to be necessary in our cases. In no instance in which it has been used has it failed to produce permanent closure of the fistula with resultant solid chest wall healing.

AVOIDANCE OF COMPLICATIONS

Complications are usually due to an inaccurate approach to the lesion. This

may result in either pleural contamination or in complications due to approaching the abscess through a portion of normal lung tissue, or both. Finding extensive pleural adhesions may lead to the false belief that one has accurately localized the abscess because adhesions may be present which bear no relation to the abscess. If one cannot obtain pus or foul air on aspiration, extreme caution should be exercised, and in most instances further *x*-ray studies are in order. Establishing a drainage tract through normal lung parenchyma opens fresh tissue to infection and increases the danger of air embolus. Our findings bear out Neuhof's[4] statement that the most superficial portion of the cavity wall is avascular. The abscess may be opened in this area without danger of air embolus.

RESULTS

The results of surgical treatment of lung abscess based upon a consecutive series of twenty-five* patients treated by the author are summarized in Table 1. There were twenty-four recoveries with one death (4.0 per cent). All patients except the one fatal case showed marked improvement as soon as adequate external drainage was provided. Postoperative empyema did not occur in any case in which one-stage drainage was used. A dissecting infection of the chest wall did not develop in any case. Cerebral air embolism was not encountered.

Five patients (Cases 11, 12, 16, 19 and 21) had two or more separate and distinct abscesses requiring separate operations. In four instances, (Cases 11, 12, 16 and 21) the lesions were in different lobes. The one fatal case had multiple abscesses in all five lobes.

The time from onset of symptoms to drainage varied from one to sixty weeks. In ten cases, the duration of symptoms was six weeks or less. The one fatal case (Case 21) was of five weeks' duration. It is quite likely he would have survived had drainage been provided before a massive spread took place. This patient illustrates the fallacy of waiting any specified time before instituting surgical treatment. One-fourth of the abscesses (six patients) followed tonsillectomy. This is a high percentage, but was assigned as the cause only when the evidence was irrefutable. Excluding the fatal case, one patient who was transferred to a psychopathic hospital, and two who are still under observation, the average period of hospitalization following drainage was forty-five days. This is in marked contrast to the duration of hospitalization on a medical or a combined medical and surgical régime. Brunn[3] reported the average duration of hospitalization in his collected series of patients under combined medical and surgical management as 154 days.

Of the abscesses facing the thoracic cage, adhesions were found in all instances when localization was correct. Note in Table 1 that the two-stage operation was supplanted by the single procedure in the more recent cases as greater attention was paid to accurate roentgenological localization. Likewise, the number of cauterizations was reduced and has approached the desired level of one per patient.

Four detailed case histories illustrating different types of abscesses encountered, including the one fatal case, are presented below.

CASE REPORTS

Case 21. Mr. J. D., Jr., a forty year old male, married, insurance salesman, was admitted to the New England Deaconess Hospital on September 26, 1940, with a chief complaint of cough, expectoration, dyspnea, palpitation and a loss of twenty-two pounds in weight.

Six weeks before admission the patient had been operated upon for chronic appendicitis under ether anesthesia. The postoperative convalescence was uneventful. However, the patient failed to regain strength. He was discharged on the twelfth postoperative day. Two days later he began to cough and raise large amounts of greenish, foul-smelling sputum, and noticed a dull ache in the left side of the chest. Blood in the sputum was seen on three occa-

*The first twenty cases in this series were included in a previous report.[7]

TABLE I

SUMMARY OF CASES

Series No.	Patient	Hosp. Adm. Date	Age	Sex	Duration Symptoms Preop. in Weeks	Etiology	*X*-ray Examination: Cavity Location: Present	Multilocular	Single Lobe	More than One Lobe	Plus interlobar Empyema	Abscess Pointing on Chest Wall, Fissure, Mediastinum or Diaphragm	Abscess Plus General Empyema	Bacteriology: Aerobic	Bacteriology: Anaerobic	Type of Operation	No of Cauterizations	P-O Empyema	Hosp. Days Drainage to Discharge	Op. Death	Late Death	Comments and Present Status*
1	C. K.	5-27-38	35	M	20	Postop. T. and A	+	−	+	−	−	Chest wall	−	Staph. Albus Hemoly.	Staph. Aureus	Exp. thoracotomy with subsequent drainage	2	+	52	−	−	Was well and working when last seen 3/39. Unable to follow since.
2	O. H.	7-6-38	49	M	48	Unknown	+	+	+	−	+	Chest wall and fissure	−	No growth	.....	One stage drainage	5	−	74	−	−	Working. Has bronchial fistula without drainage. Refuses to have it closed.
3	A. B.	9-24-38	14	F	56	Postop. T and A	+	−	+	−	−	Chest wall	−	Staph. Aureus Hemoly.	.....	Two stage drainage	1	−	13	−	−	No symptoms. Attending school.
4	E. M.	10-6-38	43	F	36	Unknown	+	+	+	−	−	Chest wall	−	Hemoly. Staph. Aureus	Unclass. bacilli	Two stage drainage	2	−	56	−	−	Asymptomatic. Does own housework.
5	L. J.	11-12-38 2-25-39	22	M	12	Postop. T and A	+	−	+	−	−	Fissure	−	Staph. Aureus Hemoly.	No growth	Two stage drainage One stage drainage	1 2	+ −	26 81	−	−	Following 1st operation had recurrence of abscess. Redrained. Empyema. Prolonged hospitalization. Working full time as truck driver. Is asymptomatic.
6	P. S.	11-21-38	53	M	9	Post convulsion	+	+	+	−	−	Chest wall	−	Spirochetes and fusiforms, Staph. Aureus	Staph. Aureus—Non Hemoly. Strept.	One stage drainage	1	−	28	−	−	No. pulmonary symptoms when last seen 3/39. Mental symptoms continue.
7	T. P.	4-17-39	56	M	21	Oral sepsis	+	−	+	−	−	Chest wall	−	Spirochetes Staph. Aureus	Staph. Aureus	One stage drainage	1	−	28	−	+	Died 11/7/39 of hypertensive cardiovascular disease. Had no post-op. pulmonary symptoms.
8	R. T.	5-12-39	50	F	7	Postpneum	+	−	+	−	+	.....	+	Staph. Aureus	Staph. Aureus	Drain. empyema and abscess	0	+ Present preop.	34	−	−	Does own housework. Bronchoscopy and bronchography, 10/9/40 showed no pulmonary pathology.
9†	T. C.	8-24-39	42	M	5	Postop. appendectomy	+	+	+	−	−	Chest wall	−	Vincent's organisms. Staph. Aureus Hemoly.	Staph. Aureus	One stage drainage	..	−	40	−	−	Working. No pulmonary symptoms.
10	E. M.	8-29-39	8	M	6	Postop. T and A	+	−	+	−	−	Chest wall	−	No growth	Staph. Aur-Hemoly.	One stage drainage	1	−	20	−	−	No symptoms. Attending school.

11	H. A.	9-12-39 2-16-40	19	F	18	Postop. T and A	+ (2)	+	−	+	+	Chest wall and fissure	−	Non-Hemoly. Strept. No growth	Strep. Strep.	First stage only of two stage drainage one stage drainage Drain. interlobar empyema	− 1 1	− − −	26 21	−	−	Following 1st operation had recurrence of symptoms and abscess in lower lobe reappeared. Following 2nd drainage had uneventful convalescence. Now asymptomatic and working.
12	C. G.	10-18-39 9-2-40	38	M	36	Unknown	+	+	−	+	−	Chest wall Chest wall	+	B. Coli B. Coli	Diphtheroids Strep.	One stage drainage One stage drainage	1 1	− −	55 32	−	−	Upper abscess disappeared following drainage of lower lobe abscess. Upper lobe process reappeared. Drained. Now asymptomatic.
13	F. C.	10-28-39 1-21-40	33	M	60	Unknown	+	+	−	−	+	Chest wall and fissure	−	B. Proteus Non. Hem. Staph.	B. Proteus No growth	One stage drainage One stage drainage	1 1	− −	43 60	− −	− −	Symptoms recurred following first operation. Redrained. Now asymptomatic and working.
14	E. B.	1-9-40	58	M	5	Oral sepsis plus accident with unconsciousness	+	+	+	−	−	Chest wall	−	Staph. Aureus	No growth	One stage drainage	1	−	56	−	+	Had no pulmonary symptoms following operation. Died 5/40 of cerebral hemorrhage.
15	C. W.	1-15-40	32	M	20	Dental extraction	+	−	+	−	−	Chest wall	−	Fusiform bacilli	B. Pyocyaneus	One stage drainage	1	−	29	−	−	Asymptomatic. Working.
16	W. G.	3-14-40	12	M	3	Unknown	+ (2)	−	−	+ (2)	−	Mediastinum Chest wall	− −	..... Hem. Staph. Aureus	..... Gram neg. rods.	One stage drainage Two stage drainage	1 1	− −	31	−	−	Developed Strep. bacteriemia. Cured by chemotherapy. Now in excellent health.
17	H. C.	5-23-40 2-27-41	47	M	20	Postconvulsive	+	−	+	−	−	Chest wall	−	..... B. Coli	..... Diphtheroids Staph.	One stage drainage (Redrainage) One stage drainage	1 2	− −	7 54		−	Transferred to Psycopathic Hospital after 1st operation. Tube removed. Abscess recurred. Redrained. Still has fistula.
18	A. C.	7-20-40	52	M	3	Oral sepsis	+	+	+	−	−	Chest wall	−	B. Coli	B. Coli	One stage drainage	1	−	28	−	−	No symptoms from abscess. Has advanced pneumonoconiosis.
19	G. D.	7-29-40	38	M	6	Unknown	+ (2)	+	+	−	−	Chest wall	−	..... B. Coli	..... B. Coli	One stage drainage One stage drainage	1 1	− −	39	−	−	Diabetic. Bronchial fistula present. Working.
20	L. H.	8-28-40	28	M	12	Post. T and A	−	−	+	−	−	Chest wall	−	Diptheroids	Diphtheroids B. Coli	One stage drainage	1	−	21	−	−	Asymptomatic. Working.
21†	J. D. Jr.	9-26-40	40	M	5	Postappendectomy	+	+	−	+	−	Chest wall	−	B. Coli Strep.	No growth	One stage drainage	1	−	Died 10 days p.o.	+	−	Extensive multiple cavities in every lobe.
22†	L. C.	12-3-40	39	M	6	Unknown	+	−	+	−	−	Chest wall	−	B. Coli fusiform bacilli	Yeast organisms	One stage drainage	1	−	20	−	−	Asymptomatic. Working.
23†	V. G.	1-30-41	31	F	2	Postpneumonic	−	−	+	−	−	Chest wall	−	No growth	No growth	One stage drainage	1	−	25	−	−	Asymptomatic.
24	C. V.	4-12-41	33	F	43	Postteeth extraction	+	+	+	−	+	Interlobar fissure	−	Diphtheroids	.....	One stage drainage	1	−	....	−	−	Has bronchiectasis. Fistula still open.
25	H. M.	4-5-41	54	F	1	Postop. cholecystectomy	+	+	+	−	−	Chest wall	−	Strep. viridans B. Coli	Staph. Aureus	One stage drainage	1	−	....	....		Still has bronchial fistula.

* As of June 1, 1941.
† Case reported in detail.

sions. He consulted his physician who had a chest *x*-ray taken. (Fig. 6A.) The *x*-ray interpretation was tuberculosis. Approximately one week later the patient entered a tuberculosis sanatorium. *X*-rays taken on admission there showed an extension of the lesion. (Fig. 6B.) Repeated sputum examinations for tubercle bacilli were negative. Bronchoscopic aspiration was carried out twice with slight temporary improvement. The course in the sanatorium was progressively downhill, the patient having a high sustained temperature, profuse prostration, copious expectoration and persistent pain in the chest. When seen in consultation, immediate surgical drainage was advised. This advice was not accepted until two weeks later.

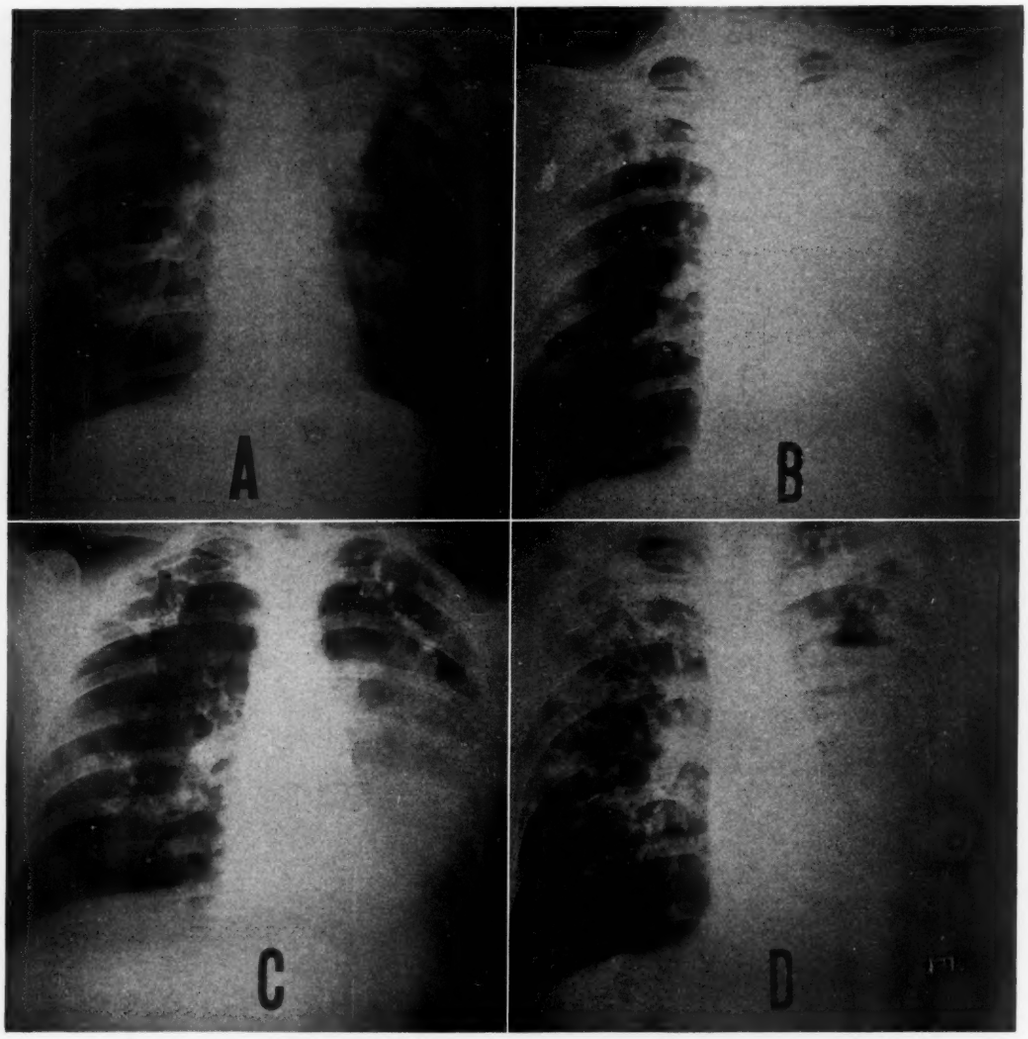

FIG. 6. Case 21. Postero-anterior *x*-rays of the chest showing rapid progress of an initially favorable lesion. A, August 29, 1940; B, September 6, 1940; C, September 25, 1940; D, September 27, 1940. Note the presence of an old tuberculous process in both apices. Due to this finding, a diagnosis of tuberculosis was made and valuable time was lost.

His past history revealed no pertinent information. The only previous illness was a gonorrheal infection at twenty years of age. There had been no known contact with tuberculosis. The patient had been married ten years. There were three children living and well.

Physical examination revealed a markedly dyspneic, emaciated, moderately cyanotic man, sitting up in bed in obvious respiratory distress. Significant abnormal findings were limited to the chest. There was dullness and diminished breath sounds over the lower two-thirds of the left chest posteriorly. Rales were heard over the entire left chest and a moderate number throughout the right chest. The heart was within normal limits and the sounds were of

good quality. The blood pressure was 88/50 mm. of mercury.

A urinalysis was essentially negative except for a very slight trace of albumin. The white blood count was 37,300, red blood count 4,180,000 per c.mm. The hemoglobin was 81 per cent. Differential: polymorphonuclears 77 per cent, lymphocytes 11 per cent, large mononuclears 8 per cent, eosinophiles 3 per cent, basophiles 1 per cent. There was moderate anisocytosis.

Roentgenographic examination (Fig. 6D) showed an extensive bilateral involvement with a large multilocular cavity of the left upper lobe. The lower two-thirds of the left chest was obliterated by a moderately dense homogeneous shadow. There was evidence of many smaller cavities throughout the upper two-thirds of the right lung.

Intensive sulfanilamide therapy was started. Although it was thought that the outlook was practically hopeless drainage of the large abscess in the left upper lobe was decided upon. The preoperative temperature was 102°F. and the pulse rate was 160 per minute. A one-stage cautery drainage was carried out on September 28, 1940. This was followed by two indirect blood transfusions.

His course following operation did not change greatly. His temperature remained quite steady at around 102°F. by rectum, and the pulse 130 to 140 beats per minute. Chemotherapy was maintained with a sulfanilamide blood level ranging from 8 to 10 mg. per cent. Fluids were administered parenterally to maintain an intake of 3,000 to 4,000 cc. per day. Blood transfusions of 500 cc. each were given on September 29, October 1, and October 4, 1940. In spite of the supportive therapy it was obvious that the patient was slowly failing but there was no dramatic change in his course until the morning of the tenth postoperative day when he became comotose, temperature rose to 104°F. by rectum, and the patient expired.

Postmortem examination revealed a healed bilateral apical tuberculous process. The lung abscess of the left upper lobe which had been drained was found to involve approximately one-half of the upper lobe. The remainder of the left lung was described as "involved by a massive destructive process." One 10 cm. abscess and many smaller abscesses were found in the right lung. A very small abscess was present around the appendiceal stump.

How well this case illustrates the rapid progress such a process may make! The issue was confused in this particular instance because of the presence of an old tuberculous process at the apices. The development of the lung abscess in the upper lobe made it seem all the more likely that it was tuberculous. This assumption accounted for part of the loss of time in the application of proper therapy. Only within the first ten days after onset of symptoms could this patient be classed as at all hopeful. By the time of admission to the sanatorium, the prognosis was very grave, again illustrative of the fallacy of temporizing with a lesion so potentially dangerous. In the light of other cases it seems quite probable that had the original abscess been drained when it was first detected, the fatal issue would have been averted.

Case 9. Mr. T. C., a forty-three year old, married, Italian clerk was admitted to the New England Deaconess Hospital on August 24, 1939, with a chief complaint of cough, expectoration, and pain in the right chest of four weeks' duration.

On August 16, 1939, the patient developed symptoms of acute appendicitis. At operation the following morning a ruptured appendix was found. This was removed and a drain inserted. A right inguinal hernia was repaired at the same time. The patient had a stormy time postoperatively and ten days after operation began to complain of pain in the left chest. A cough developed and the temperature became more elevated. It was thought at first that he had a pulmonary embolus. Soon the patient began to raise large amounts of foul-smelling material. A fecal fistula developed at the site of the appendectomy. Sulfapyridine was given without benefit. Roentgenological examination of the chest revealed a large lung abscess in the left lower lobe. Thoracic surgical consultation was requested and the patient was transferred the following day to the New England Deaconess Hospital.

Physical examination revealed a very ill man sitting upright in bed, breathing with effort, coughing frequently and raising foul sputum. Many rales could be heard throughout the posterior aspect of the left chest. Amphoric

breath sounds were audible over the seventh and eighth ribs posteriorly. A profusely draining sinus was found in the right lower quadrant of the abdomen. A right inguinal herniorrhaphy scar was well healed. The remainder of the examination was not significant.

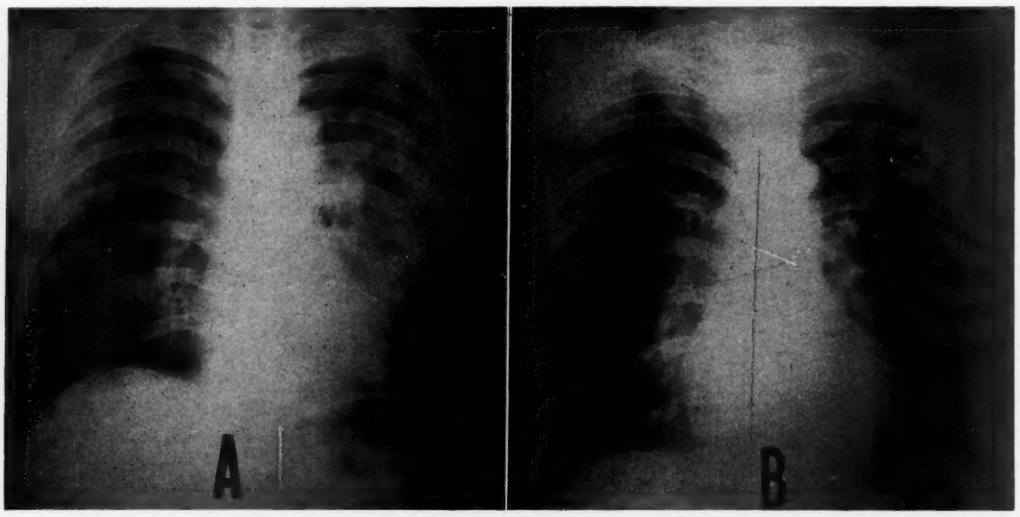

Fig. 7. Case 9. Postero-anterior *x*-rays of chest. A, August 24, 1939. Note large multilocular abscess with surrounding pneumonitis in left lower lobe. B, August 23, 1940. Lesion has now cleared completely, following surgical drainage, except for slight pulmonary fibrosis.

Urine examination was normal except for 14 to 18 white blood cells per high power field. The white blood count was 21,700; red blood count, 3,580,000 per c.mm. The hemoglobin was 50 per cent. Differential: polymorphonuclears 92 per cent, lymphocytes 3 per cent, eosinophiles 1 per cent, young polys 4 per cent. Moderate achromia of the red blood cells was noted.

X-ray examination of the chest (Fig. 7A) revealed a large multilocular abscess in the left lower lobe with surrounding pneumonitis.

The patient's condition was desperate, temperature 101°F., pulse rate 146 per minute. A blood transfusion of 500 cc. was given as soon as possible after admission, and later that day a one-stage cautery drainage of the lung abscess of the left lower lobe was carried out.

The patient's improvement following operation was dramatic. The temperature returned to normal by the end of the first week. The pulse rate gradually dropped to an average of 100 within the same time. The fecal fistula closed spontaneously. The patient was able to be out of bed thirty days after operation and was discharged seven days later. The large defect of the left lower lobe gradually filled in and shortly before discharge a drainage tube was inserted to keep the skin from closing over. The patient was followed at weekly intervals. For some time it was thought that it would be necessary to do a plastic operation in order to close the bronchial fistula. However, the fistula healed spontaneously and the tube was removed on January 2, 1940, three and one-half months after discharge from the hospital. Since that time he has been entirely without symptoms, now one and one-half years. He has returned to full time work and is in excellent health. (Fig. 7B.)

The postoperative response of this patient presents a dramatic illustration of the marked improvement that may be experienced by an acutely ill, toxic patient as soon as the abscess is decompressed and the patient is freed of persistent coughing and raising. Although this particular case was complicated due to an extensive pulmonary process with surrounding pneumonitis, a fecal fistula, and marked debilitation by the long septic process, the period of hospitalization following operation was not excessive and an excellent result was obtained. Judging from the patient's preoperative course, further temporizing would have been fatal.

Case 22. Mr. L. C., a thirty-nine year old, married, American, carpenter was admitted to the New England Deaconess Hospital December 3, 1940, with a chief complaint of cough and expectoration of six weeks' duration.

The patient stated that in March, 1939, he was in another hospital for eight days because of cough and the raising of profuse, foul-smelling sputum. A diagnosis of lung abscess was said to have been made at that time on *x*-ray and clinical evidence. Upon discharge from the hospital he remained home for four months on a modified rest program. He then returned to work.

He felt well until the latter part of October, 1940, when he suddenly felt severe pain in the left side of the chest. After a two weeks' period at home he was transferred to the Essex County Tuberculosis Sanatorium for observation. Although numerous *x*-rays had been taken previously, a film on November 7, 1940, (Fig. 8A) was said to have been the first one to show any abnormality. The cough was quite distressing and he raised from two to three ounces of foul sputum per day. Surgical drainage was advised.

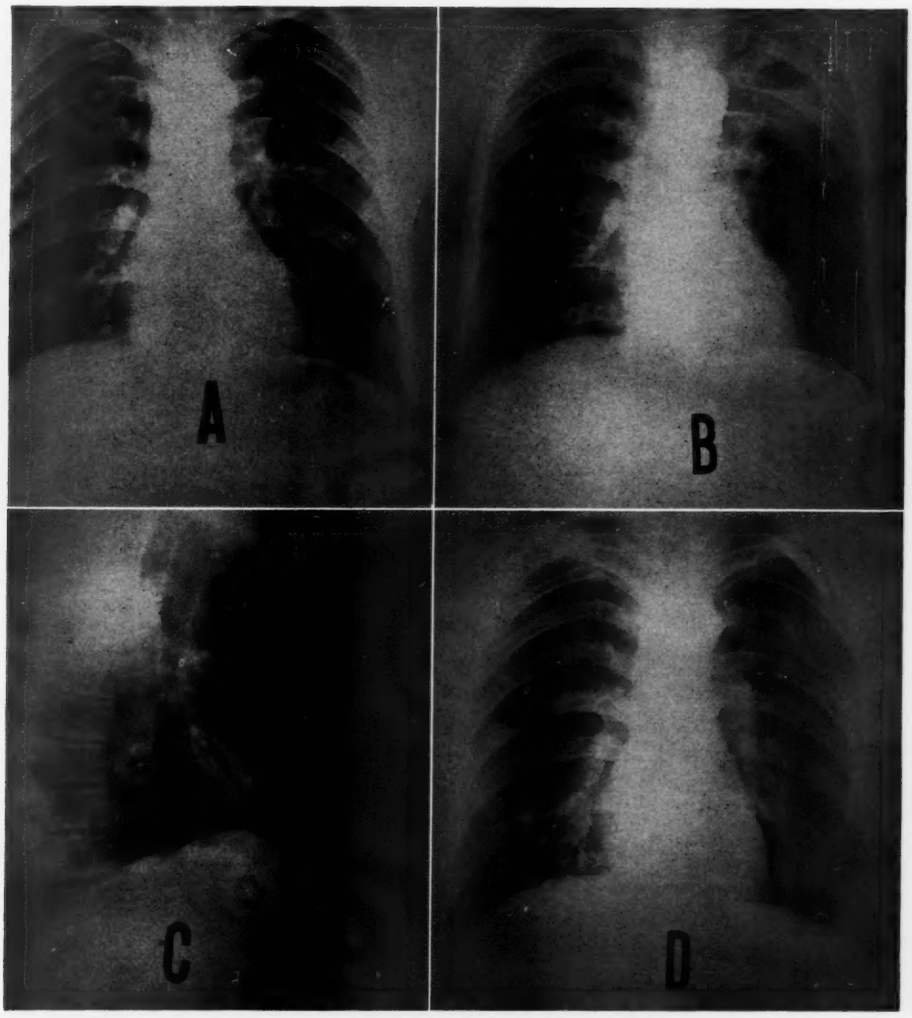

Fig. 8. Case 22. A, postero-anterior *x*-ray of chest. November 7, 1940, showing some infiltration of mediastinal segment of left upper lobe. The only complaint at that time was severe chest pain. B and C, postero-anterior and left lateral *x*-rays of chest approximately one month later, December 4, 1940. Patient now has a large lung abscess with marked cough and profuse expectoration. D, February 10, 1941. Lesion has now cleared completely following surgical drainage. Note lipiodal still in intercostal tissue above resected rib.

His past history was negative except for the episode recorded in the present illness. The family and marital histories were noncontributory.

Physical examination revealed the patient to be a well developed and well nourished man in no particular distress. There was moderate oral sepsis. The chest was symmetrical and expansion was full and equal. There was dullness to percussion over the upper part of the left

posterior chest. Harsh breath sounds were noted together with moist rales in this same area. The remainder of the chest showed nothing abnormal. The abdomen showed a well healed lower quadrant scar. Extremities were negative.

Laboratory examination revealed nothing abnormal in the urine. The white blood count was 28,200; red blood count, 4,650,000 per c. mm. and the hemoglobin was 87 per cent. Differential: polymorphonuclears 87 per cent, lymphocytes 5 per cent, large mononuclears 4 per cent, eosinophiles 4 per cent, basophiles 1 per cent.

Roentgenological examination of the chest (Fig. 8B and C) revealed an abscess in the lower part of the left upper lobe. A one-stage cautery drainage of the lung abscess was carried out December 4, 1940.

Postoperative convalescence was entirely uneventful, the temperature never going above 100°F. The pack was first changed four days after operation and every other day thereafter until the seventeenth postoperative day when a small rubber tube was inserted in place of the pack. The patient was allowed out of bed fifteen days after operation and was discharged on the twentieth postoperative day. Following discharge from the hospital, he was followed at weekly intervals and on February 1, 1941, the fistula was found to be closed and the tube was therefore removed. The chest wall healed promptly. When last seen on March 24, 1941, the patient had no complaints and an *x*-ray (Fig. 8D) showed complete clearing of the pulmonary process. He was given permission to resume normal activities.

One can speculate a great deal as to the length of time the lesion had been present. It is entirely possible that the recent abscess was the same lesion that was present in March, 1939. The re-activation of an abscess after apparent spontaneous healing has been a rather frequent occurrence. The rapid recovery made in this case again demonstrates that a smooth convalescence can be expected following drainage of an uncomplicated single abscess. Recurrence is rare following external drainage.

Case 23. Miss V. G., a thirty year old, single woman, was admitted to the New England Deaconess Hospital on January 30, 1941, with a chief complaint of cough and temperature of two weeks' duration. Two weeks previously the patient had suddenly developed a high temperature, cough, and pain in the right chest. She was admitted to the Joseph H. Pratt Diagnostic Hospital where a diagnosis of right upper lobar pneumonia was made. A chest *x*-ray revealed a shadow in the right upper lobe consistent with lobar pneumonia. Intensive chemotherapy was given. Her temperature continued to range from 100 to 102°F., pulse rate 100 to 120. After thoracic surgical consultation, surgical drainage was advised and the patient was transferred to the New England Deaconess Hospital.

The patient had been a quite severe asthmatic since August, 1938. She was known to be sensitive to dust, lettuce and spinach. On intensive allergic therapy, however, she had been able to control her symptoms quite well. Two years previously a splenectomy had been performed for a ruptured spleen following an automobile accident. One year previously an ectopic tubal pregnancy was removed.

Physical examination revealed a thirty year old white woman sitting upright in bed in obvious respiratory distress. Respiratory wheezing was marked. Examination of the head and neck showed no abnormality except for very poor teeth. Percussion of the chest revealed dullness and diminished breath sounds over the upper part of the right chest posteriorly and in the right axilla. No rales were audible. Tactile and vocal fremitus were slightly increased. Wheezing and prolonged expiratory sonorous rales were heard throughout both chests. The heart appeared to be of normal size and without murmurs. The blood pressure was 120/70 mm. of mercury. Abdominal examination was negative except for left upper quadrant and low midline well healed scars. The remainder of the physical examination showed no abnormality. The temperature was 102°F.; pulse 140; respirations 30.

A urinalysis showed no marked abnormality except a slight trace of albumin and an occasional fine granular cast. The white blood count was 19,400; red blood count 4,600,000; hemoglobin 70 per cent. The differential count showed 63 per cent polymorphonuclears, 15 per cent bands, 7 per cent lymphocytes, 10 per cent monocytes, and 5 per cent eosinophiles. Serological examinations were negative. One blood culture was sterile.

Roentgenographic examination of the chest (Fig. 9A and B) revealed evidence of consolidation of the posterior portion of the right upper lobe. The shadow was limited below by the interlobar fissure, but the upper borderline was indistinct. No definite cavity was visible. Although definite evidence of a lung abscess by *x*-ray was not present and the patient had practically no sputum, a diagnosis of lung abscess or interlobar empyema was made and operation advised.

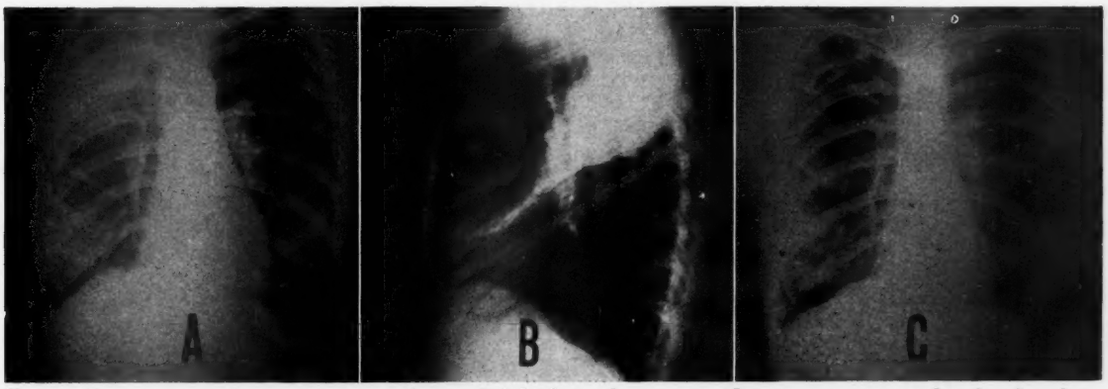

Fig. 9. Case 23. A and B, postero-anterior and right lateral *x*-rays on January 29, 1941, showing involvement of lower part of right upper lobe. No evidence of cavitation. C, posterior-anterior *x*-ray April 18, 1941. Note complete clearing of process following surgical drainage.

On January 30, 1941, (the day of admission to the Deaconess Hospital), under local anesthesia, the posterior aspect of the right upper lobe was explored after resecting a portion of the fifth rib posteriorly. A multilocular abscess was entered, the cavities being filled with very thick, cheesy, yellowish pus. These abscesses were broken down and the space packed with gauze after application of sulfathiazole powder. Bacteriological examination of the material from the abscess revealed sterile cultures both aerobically and anaerobically, although Gram-positive cocci were found in clumps upon direct smear.

The patient's postoperative convalescence was satisfactory. Although the temperature rose to 103.5°F. the day of operation, it returned to normal quite rapidly and her general condition improved markedly. The patient was discharged from the hospital twenty-five days following drainage of the abscess.

She was followed at weekly intervals. A small tube which had been in place in the sinus tract before she left the hospital was withdrawn as the sinus tract closed in. The tube was removed on May 16, 1941. She has been seen on two occasions since that time. The wound is completely healed, and the last *x*-ray (Fig. 9C) shows complete clearing of the lesion.

In this case a persistent area of infiltration by *x*-ray and continued signs of sepsis following an attack of pneumonia suggested pulmonary suppuration. Destruction of pulmonary parenchyma did not seem to be as extensive as that usually caused by anaerobic organisms. No area of excavation was present roentgenographically but at operation many small connecting pockets were found. It is better to expose such lesions surgically than to wait a prolonged period of time for definite evidence of cavitation by *x*-ray.

SUMMARY AND CONCLUSIONS

Early drainage of lung abscess has yielded a low mortality rate and a high cure rate in our experience. A series of twenty-five consecutive cases of pulmonary abscess treated by surgical drainage has been presented. There was one operative death.

Inhalation of material from the upper respiratory passages appeared to be the causative factor in the vast majority of cases. Symptoms had been present from one week to one year and two months before operation. Five patients had multiple abscesses, four of which were in more than one lobe.

In all cases surgical drainage was provided as soon after admission to our service

as diagnostic and localizing studies could be completed. Critically ill patients were treated as surgical emergencies.

Early surgical drainage protects the patient from the danger of bronchiogenic spread, multiple abscess formation and extensive irreparable pulmonary damage.

Pulmonary abscess should be considered a surgical disease from its onset. Every effort should be made to establish the diagnosis, localize the lesion, and provide external drainage without delay.

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RECOGNITION AND MANAGEMENT OF MEDIASTINITIS*

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ALTHOUGH chronic mediastinitis is a rare disease, acute mediastinitis is not nearly so infrequent as the profession once believed. Numerous reports in the past few years indicate the fairly common occurrence of acute infections of the cervical and thoracic mediastinum. When one considers the ease of access to the fascial planes for spontaneous infections of the mouth, nose and throat, cervical lymph-nodes, neoplasms of the pharynx and esophagus, and foreign bodies, it is fortunate that nature is so kind in building inflammatory barriers to prevent the downward extension of many of these infections.

An accurate concept of the practical anatomy of these fascial planes of the neck and thorax, as so vividly described by Furstenberg and Yglesias, demonstrates the continuity of the retrovisceral space as a compartment (Figs. 1 and 2) bounded behind by the prevertebral layer of cervical fascia, in front by the pharynx and esophagus and the lateral extension of the retroesophageal fascia, which is a derivative of the pretracheal layer, and laterally by the carotid sheaths. It extends from the base of the skull to the diaphragm. This makes easy migration of infection from the retropharyngeal or retroesophageal locations into the posterior thoracic mediastinum, and it is remarkable that inflammatory barriers so frequently localize the infection to the cervical portion. Surrounding the viscera of the neck consisting of the pharynx and esophagus behind and trachea and thyroid in front, is a cylindrical sheath. This consists of the pretracheal layer which extends laterally to join the prevertebral layer at the carotid sheath and is continued posteriorly as the retropharyngeal or buccopharyngeal fascia above and the retroesophageal fascia below, constituting the vasculovisceral fascia. This blends with the aorta as the arch crosses the trachea and encloses the descending limb. It continues downward to fuse with the pericardium at about the level of the bifurcation of the trachea at the sixth thoracic vertebra. Thus the other practical mediastinal space, the anterior or visceral space, extends from pharynx and larynx above to the bifurcation of the trachea below. The potential space immediately behind the sternum, included in anatomy textbooks as a part of the anterior mediastinum, is protected from the spread of cervical infections by the junction of the superficial layer of cervical fascia and its muscular investments, with the posterior surface of the manubrium sterni so that infections reach this space only when they originate from injury or infection of the anterior chest wall at the level of the first and second costal cartilages.

In the vasculovisceral fascial compartment lie many lymph-nodes and vessels, and it is into this space that nose, throat and ear infections frequently travel. On the other hand, perforations of the posterior pharyngeal or esophageal walls by foreign bodies usually involve the retrovisceral space and are more prone to extend deep into the thorax. One or both of these spaces may be implicated and the intimate relationship to the pleurae and pericardium leads to infection of these at times. Practically, then, there are two main spaces, the retrovisceral and the vasculovisceral, which may be infected and are continuous from neck to thorax with only theoretical, but no actual division between the cervical and thoracic portions. The concept that the mediastinum extends from the base of the skull to the diaphragm is a good one for practical purposes.

The extent to which infection of these spaces may spread is influenced by the

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amount of contamination, the virulence and the frequent multiplicity of organisms on the one hand, and the patient's resistance on the other. Thus a bad combination of perforation by foreign body through a small opening which does not allow spontaneous drainage, in a patient who has streptococci and spirochetes from oral sepsis and is suffering from more or less nutritional deficiency, may frequently lead to a rapidly spreading, diffuse, and necrotizing plegmon with such extreme absorption and toxemia as to result in death in one or two days. For such a patient, the prospects are poor, even with early drainage. Conversely, in patients with good resistance who have sustained perforations of the pharynx or esophagus with the demonstration of gas in the tissues, but in whom virulent bacteria were not present in large numbers, spontaneous and fairly rapid recovery has occurred on a few occasions. Between these two lie the great majority of cases resulting from either infections about the mouth, ears, or throat, or trauma to the pharynx and esophagus in which all combinations of the above two extremes occur. In many of these the infection is localized by inflammatory barriers and retained in the cervical region as a retropharyngeal abscess for a longer or shorter time. Even these, if neglected, tend to spread downward in many instances.

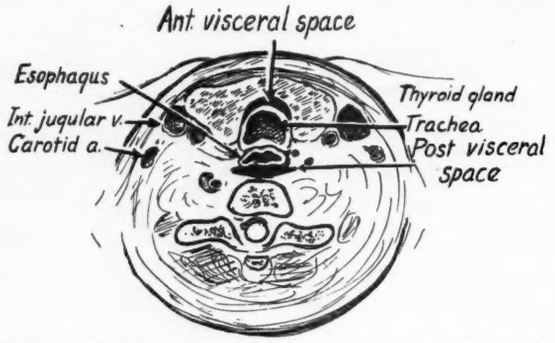

Fig. 1. Diagram of coronal section at the level of the sixth cervical vertebra.

In a review of twenty-eight cases of cervical and thoracic infection of the mediastinum and ten cases classed as retropharyngeal abscess who were admitted to all services of Temple University Hospital over a period of the past five years, the source of infection is indicated in Table 1. The most common causes were perforation by foreign body and neoplasm and infections of the mouth and throat. Three of these latter followed extraction of teeth. One case occurred after otitis media and six cases after diagnostic instrumentation in the presence of ulcerative lesions, some of whom had symptoms suggestive of infection before examination. Two cases followed laryngectomy, and one thyroidectomy for extensive malignant disease. One patient sustained spontaneous perforation of the esophagus. Of the abscesses classed as retropharyngeal, eight followed upper respiratory infection, in most of which sore throat was stated, and two were secondary to otitis media.

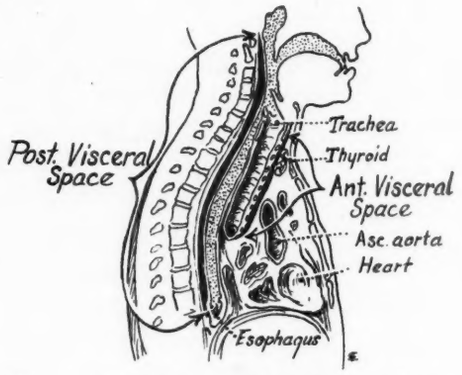

Fig. 2. Diagram of midsagittal section.

Table 1
Origin of Mediastinitis

	No.
Foreign body	9
Carcinoma esophagus	4
Perforation esophagus	1
Infections	6
Otitis media	1
Upper respiratory	2
Ludwig's angina after tooth extraction	3
Following endoscopy	4
Following operation	3
Laryngectomy	2
Thyroidectomy	1
Following passage of feeding tube	1
	28

The symptoms of the twenty-eight cases (excluding retropharyngeal abscess): Eleven showed dysphagia, eight dyspnea—

two of whom required tracheotomy—six had pain in the throat and seven pain in the chest, neck or back. In four there was vomiting, in three hemoptysis, and in two there was hoarseness. All but two had fever and in twenty it was moderate to high and frequently septic in character. Chills occurred in only three cases. Cyanosis was present in seven, swelling of the neck in thirteen and cervical tenderness in twelve. Abnormal chest signs were present in four cases.

The most constant evidence was demonstrated by *x*-ray. Due to obvious diagnosis or failure to consider the possibility, no *x*-ray was taken in four cases, but in the remainder all showed widening of the mediastinum or of the retrotracheal soft tissues depending upon location, and in fourteen there was gas present in the tissues.

Of the so-called "retropharyngeal" group, the symptoms consisted rather constantly of sore throat and difficulty in swallowing. A few references were made to labored or noisy breathing and to swelling of the neck. In the occasional case in which roentgen examination was done, thickening of the space between the vertebra and pharynx was revealed.

A review of the multiple cases in the literature shows that the etiology, symptoms and signs ascribed to the patients reported correspond to these we have outlined as to frequency of occurrence. The rare circumstance of mediastinitis secondary to osteomyelitis of the thoracic vertebra, suppurative lymphadenitis of peribronchial or peitracheal lymph-nodes from perforation or operation of neoplasms of the thoracic esophagus, and following pulmonary suppuration, empyema, or thoracic operations, is reported. Such origins usually result in abscesses localized to the thoracic portion of the mediastinum and may not be accessible from the cervical approach but require posterior mediastinotomy.

Acute mediastinitis is susceptible to accurate diagnosis if one will only consider the possibility of its occurrence with infection or trauma of the mouth, nose, throat, ears or esophagus. Any patient may be considered a candidate for such diagnosis if any one or combination of the following is present: difficulty in breathing or swallowing, pain in the throat, tenderness or swelling in the neck. The only acute conditions which are likely to confuse one clinically are acute lymphadenitis, possibly tuberculous adenitis and the rare conditions causing increased superior vena caval pressure. The finding of emphysema in the cervical or thoracic mediastinum by clinical or *x*-ray means and the thickening of the visceral and retrovisceral spaces or widening of the mediastinum by *x*-ray associated with fever and leukocytosis, clinch the diagnosis. The main factors are the consideration of the condition in potential cases and the examination by *x*-ray of these soft tissues.

Results. In our group of ten cases in which drainage was not established surgically, four recovered and six died. Of the eighteen cases in which drainage was established surgically, twelve recovered and six died. These last six deaths were in patients with thoracic mediastinal involvement. The causes of death ascribed are tabulated. (Table II.) Thus the mortality was 33⅓ per cent of patients in whom surgical drainage was established and 60 per cent for those in whom it was not. (Table III.) Delay before admission, before recognition, and before surgery contributed significantly to the mortality of both groups since there was no intention to deny surgery to those patients. In only three of the twelve fatal cases did it appear that the virulence and extent were too great for surgical salvage, although it is questionable in two others even if early recognition and drainage had been accomplished. Although a few cases recover without operation, it is difficult if not impossible to select these in the early stages and it seems extremely dangerous to delay surgical drainage because of the occasional nonsurgical recovery. Delay not only increases the

parenchymatous damage from prolonged toxemia, but frequently leads to spread of an otherwise circumscribed infection. Particularly with the perforations of the pharynx and esophagus, constant recontamination occurs unless an adequate external vent is produced. The ten cases classed as retropharyngeal abscess were drained early through the posterior pharyngeal wall with rapid recovery.

TABLE II
CAUSES OF DEATH

Case	Days from Onset to Drainage	With Surgical Drainage
1	9	Carotid hemorrhage twelve days postoperative—ligation of carotid—hemiplegia—died in twenty-four hours
2	12	Delay—moribund at operation—edema—thin cloudy fluid
3	43	Died suddenly two days postoperatively—vascular accident
4	8	Inadequate drainage—temporary improvement—readmitted and died seven weeks later with healed wound and continued mediastinitis
5	8	Followed laryngectomy—extensive sloughing of wound
6	7	Followed thyroidectomy for advanced malignancy—extensive sloughing of wound and metastases
		Without Surgical Drainage
1	17 hrs.	Too ill for multiple surgery—pericardial and bilateral pleural infection
2	4	Error in diagnosis—empyema only was drained
3	10	Moribund—error in diagnosis—only superficial extensions to neck and shoulder were drained
4	4	Unrecognized until postmortem examination; pericardial and bilateral pleural infection
5	8	Delay—moribund on admission—died during diagnosis in eighteen hours
6	30	Error in diagnosis

MANAGEMENT

When the diagnosis is established, cervical mediastinotomy should be instituted as an emergency procedure in every case with the exception of those rare infections which begin and remain below the fourth rib posteriorly. With such rare exceptions, posterior mediastinotomy should be done by resecting portions of two or three ribs and transverse processes at the appropriate level and retracting the pleura laterally and anteriorly until the mediastinal collection is entered. Soft rubber tubes, rubber dam or gauze are inserted to maintain the opening, and irrigations with oxidizing solutions are utilized until the drains can be gradually shortened and withdrawn. No posterior mediastinotomies were employed in this group, although indicated in Case XXIII.

TABLE III
197 CASES FROM THE LITERATURE

	Operated		Nonoperative	
	Recovered	Died	Recovered	Died
Pearse	44	24	6	36
99 collected				
11 personal				
Phillips	17	3		
Neuhof	25	7	9	25
	86	34 (39.5 per cent)	15	61 (80 per cent)

Cervical mediastinotomy should be instituted on the side of the neck which is indicated by clinical or radiological localization, or upon the right side if the infection is bilateral or only posterior. There is less likelihood of entering the pleura on this side than on the left. An oblique or transverse incision is made along the anterior border of the sternomastoid muscle through the superficial layer of the deep cervical fascia. The sternomastoid and carotid sheath are retracted laterally and the thyroid retracted medially which usually entails division between ligatures of the lateral vein and at times the inferior thyroid artery. If the abscess is anterior, it will be found more medial under the pretracheal fascia and if posterior, directly backward through the dense union of the pretracheal and retroesophageal fascial

planes with the carotid sheath. If palpation reveals a large space extending to the opposite side, some surgeons advocate repeating this procedure on the opposite side, although usually a unilateral approach allows adequate drainage.

If foreign body or instrumental laceration is suspected, search should be made for it. Foreign bodies can frequently be removed by esophagotomy alone or combined with esophagoscopy. Lacerations or surgical wounds have a marked tendency to reopen after suture unless fine steel wire is used. If adequate drainage is established, such openings heal spontaneously. With the finger, one must explore cephalically and caudally to determine the extent of the abscess and place two small rubber tubes at the extremities in each direction. Two tubes give better drainage than one, both through the lumens and between the pair, and allow for irrigation of deep spaces without production of dangerous positive pressure. Constant irrigation by drip with oxidizing solution such as Dakin's or azochloramid, offer the best chance for rapid dilution and mechanical removal of infectious products. One should avoid too great or too prolonged pressure on the carotid or jugular vein because of the danger of serious hemorrhage.

When the abscess involves the thoracic portion of mediastinum, postural drainage by modified Trendelenburg position is important to assist drainage by gravity.

To overcome dysphagia and promote rest of this area, and particularly if the esophagus is the source of contamination, a feeding tube should be inserted into the stomach at the time of operation so that it can be guided beyond the laceration by the operator. Of particular value here is the double barrel tube such as the Abbott-Rawson through one channel of which feeding can be carried on while the esophageal secretions are constantly aspirated through the other to lessen recontamination by the swallowed saliva. Of course, nothing is given by mouth for the same reason. Nutrition, fluid and chemical balance must be maintained by feeding tube or intravenous administration.

Additional therapy such as the sulfonamide drugs in adequate dosage, neoarsphenamine if mouth organisms enter the picture, and frequent small blood or plasma transfusions to activate complement and combat anemia and nutritional disturbances help to limit further spread and to hasten cure.

Progress is gauged by clinical means, but must include check-up blood count and *x*-ray examinations to avoid too early removal of drainage with the possibility of resultant reaccumulation. Unfortunately, one of our patients died from failure to follow this schedule and to determine the extension of the abscess into the thoracic mediastinum. With adequate drainage and supportive measures, patients should recover in from two to four weeks. The pain, dyspnea and dysphagia disappear in a few days, and the temperature usually falls to normal in from five to ten days. Profuse drainage after two weeks or prolongation of more than four weeks usually means stenosis of the drainage tract with inadequate vent.

CASE REPORTS

Case I. H. C., aged fifty-nine, was admitted January 31, 1940. He complained of pain in the renal area radiating to pubes and hematemesis five days before. He felt better on an ulcer diet until the evening of the next day when bloody vomitus recurred with hoarseness and anterior swelling of the neck. On physical examination there was cyanosis, stridor, ulceration of right pyriform sinus and swelling of the neck and supraclavicular regions. Breathing was difficult but there was no increased mediatinal dullness. The day after admission a tracheotomy was performed and adequate drainage obtained by cervical mediastinotomy. *X*-ray examinations postoperatively showed esophageal perforation and retropharyngeal abscess with extension to the mediastinum. Temperature receded from 102.2°F. immediately postoperative to 98°F. in eleven days, and he was discharged on March 2 without symptoms. (Fig. 3A and B.)

Case II. G. M., aged nineteen, was admitted May 20, 1936, with the history that

eight days before he had swallowed a denture containing two teeth while eating cream pie. He did not miss the denture until he tried to say "f" or "v." After *x*-ray, attempt at removal was done in his home town followed by two further unsuccessful attempts, the last under cyclopropane. A few hours later there was hemoptysis and emphysema of the neck with temperature of 101.4°F. Two attacks of cyanosis with irregular heart action occurred in the next two days. On admission temperature was 98.3°F. and *x*-ray revealed gas in the mediastinum, both cervical and thoracic. There was no definite thickening of the retrotracheal tissues, but there was a small amount of gas in both pleural spaces. The foreign body was visualized in the neck. There had been marked regression of gas since examinations before admission. Under observation the gas entirely disappeared so that by June 1 none could be demonstrated radiologically. The temperature meanwhile ranged between 98 and 99°F. The

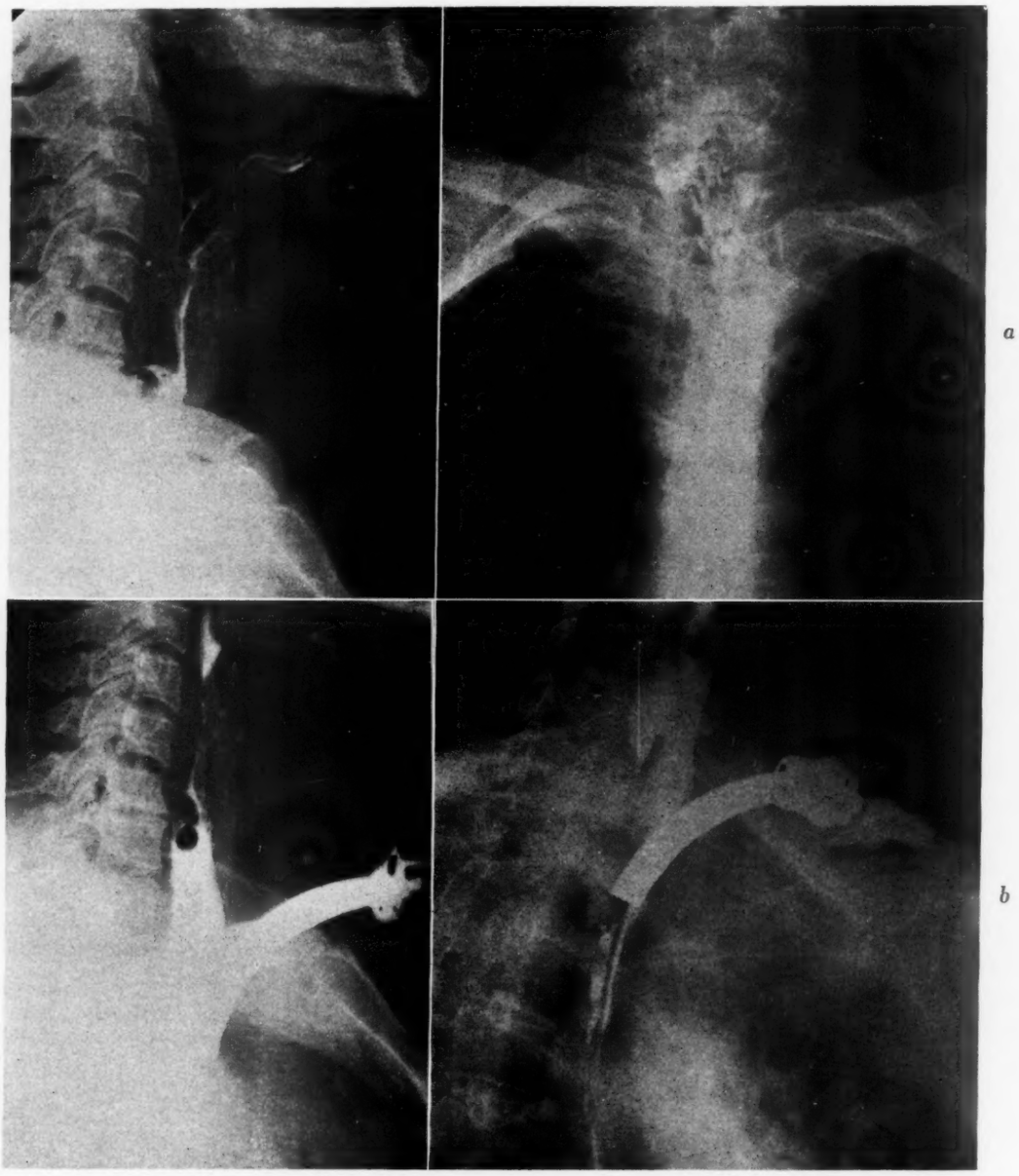

FIG. 3. *a*, Case 1. H. C., *x*-ray films of February 1, 1940, showing widening of the retrovisceral space with gas extending to the level of the fifth rib posteriorly. *b*, *x*-ray films of February 7, 1940, showing tracheal tube, decreased retrovisceral depth, and barium extending into perforation.

esophagus was observed on several occasions and when sufficient recession of inflammation had occurred an attempt was made to remove the foreign body, but it was found too badly imbedded for safe endoscopic inspection. After another such attempt, external removal by cervical mediastinotomy was done on June 29, six weeks after admission. Operation revealed no pus but there was gray purulent drainage for ten days and the temperature reached normal in twelve days. He was discharged July 15, cured.

Case III. M. K., aged sixty-nine, was admitted June 21, 1936, for dysphagia of nine weeks' duration due to carcinoma of the esophagus. Physical examination was entirely negative except for a suggestion of mass in the epigastrium. On this day he had a chill which was ascribed to intravenous glucose and the following day biopsy was obtained through the esophagoscope. On July 24, he suffered another chill and fever to 104°F. which subsided for a few days to below 100°F. There was dullness posteriorly in the intrascapular region. Four days later the temperature had continued with additional chill and a tender swelling occurred at the base of the neck with increased width of mediastinal dullness. This increased and on August 3, superficial drainage of a necrotizing infection of the sternomastoid, scalenus, and trapezius muscles was instituted, but the mediastinum was not entered. He died the following day, August 4, and postmortem examination revealed a spontaneous perforation through the esophageal carcinoma with pus in the visceral and retrovisceral spaces.

Case IV. J. C., aged sixty-four, was admitted January 17, 1938, with hoarseness of two months' duration due to carcinoma of the larynx. On January 20, laryngectomy was done and by January 31, the wound had broken down with considerable sloughing and exposure of infection in the pretracheal tissues. Multiple débridement and attempts at closure of the esophageal fistula were done until April 14, when a portion of necrotic sternum was excised and better drainage of the mediastinum was established. *X*-ray examination on March 8 revealed widening of the superior mediastinum, a horizontal streak of density above the left diaphragm, and elevation of both leaves of the diaphragm. He was discharged on June 9, with continued drainage, most of which was due to esophageal fistula.

Case V. F. W., aged fifty-nine, was admitted August 17, 1939, for hoarseness of two and one-half years due to carcinoma of the larynx. On August 24, laryngectomy was done and three days later the temperature rose to 104°F. and remained elevated. Drainage was instituted and on September 1 the wound was opened widely for adequate drainage revealing involvement of the peritracheal tissues in the mediastinum. His temperature continued to rise and he died on September 4. No postmortem examination was obtained.

Case VI. L. B., aged seventy-two, was admitted January 17, 1937, with fairly extensive carcinoma of the thyroid (microscopically giant cell). On January 19, tracheotomy and thyroidectomy were done. His temperature gradually rose until January 26 when he was dyspneic, had auricular fibrillation, and a foul sloughing wound communicating with the anterior mediastinum. On January 28, *x*-ray revealed multiple pulmonary metastases. He became progressively worse and died on February 9. Postmortem examination revealed residual carcinoma in the neck, multiple pulmonary metastases, mediastinitis, right empyema and a terminal bronchopneumonia.

Case VII. G. McD., aged six, was admitted January 14, 1939, with a history of sore throat and stiffness in the neck beginning five days before, and followed on the next day by swelling of the right side of the neck. Two days before her temperature had gone to 104°F., there was vomiting, and the following day dyspnea, cough and pain in the left lower chest. On examination she was cyanotic, dyspneic and there was a tender fullness in the neck anteriorly. The left chest revealed restricted movement, dullness and diminished breath sounds. Her temperature was 102°F. and respiration 44. *X*-ray revealed mediastinal widening and fluid in the left pleura. She was given prontolin intravenously and sulfapyridine by mouth and her blood concentration varied between 2 and 13 mg. per cent. Fluid was aspirated from the pleura on January 16, 17, and 21, and she was given five transfusions. Cultures of pleural fluid showed Streptococcus hemolyticus. Medication was discontinued on February 12, four weeks after admission because the patient had apparently been cured. She was discharged as cured on February 16 with negative physical examination and *x*-ray evidence of slight limitation

of diaphragmatic movement but otherwise negative.

Case VIII. M. C., aged five, was admitted August 17, 1938, for bilateral otitis media and right mastoiditis of three weeks' duration. On August 29, there was considerable nasal discharge, although the ears and mastoid had improved markedly. She developed cough, grunting respiration, and cyanosis and there were abnormal physical signs in the left upper lobe area. *X*-ray revealed tracheobronchial lymph-node enlargement with obstructive emphysema. This later progressed to atelectasis and by September 6, the left lower lobe was also airless. Several aspirations were unsuccessful in producing fluid, and bronchoscopy revealed huge quantities of pus from the left bronchus containing hemolytic streptococci and fusiform bacilli. The patient was treated by blood transfusion, sulfonamide drugs and neo-arsphenamine, but progressed to death on September 29. *X*-ray had revealed diminution in the mediastinal width as the suppurative pneumonitis progressed, which was interpreted in retrospect as being due to drainage of the mediastinitis spontaneously into the left upper lobe. The *x*-ray diagnosis of mediastinal lymph adenopathy and a positive Mantoux test had led to the erroneous diagnosis stated above. No postmortem examination was obtained.

Case IX. A. B., aged twenty, was admitted May 6, 1938, with a toxic psychosis for which tube feedings were begun. The patient withdrew the tube on two occasions and on May 8, there was considerable difficulty and struggle in reinserting it. The following day, May 9, there was emphysema of the face, neck, thorax and arms. Temperature was 104°F., pulse 160, respiration 20, white blood cells 17,750 with 88 per cent polymorphonuclear neutrophils and of these 38 were nonfilament forms. He was placed on nothing by mouth, sulfanilamide, 100 gr. daily, and daily small blood transfusions. *X*-ray revealed emphysema but no thickening of the cervical and thoracic mediastinal tissues. There was rapid improvement, the temperature reaching normal in six days, although there were two subsequent rises from May 17 to 21 and from May 23 to June 3, as high as 104°F. on one day, but *x*-ray revealed no thickening and decreased gas in the mediastinum. Observation over the next three months during which the psychosis improved, showed no further evidence of mediastinal infection.

Case X. T. C., aged fifty-six, was admitted December 11, 1937, with a three-month history of dysphagia due to carcinoma of the esophagus. Endoscopic examinations were made on December 13 and 16 without difficulty and the constriction visualized. On December 17, temperature rose to 100°F. and there was swelling and tenderness on the left side of the neck along the anterior border of the sternomastoid. This was drained and 5 to 10 cc. of thin odorless pus evacuated, which upon culture was sterile. Seven days later a gastrostomy was done and the patient was discharged on January 15, 1938, with no further evidence of mediastinal infection.

Case XI. S. W., aged seventy, was admitted February 15, 1938, with a history that she had suffered dysphagia progressively for six weeks and substernal pain for the past two or three weeks. She had lost twenty pounds in weight. Examination revealed temperature of 101°F. with swelling and tenderness of the left side of the neck. White blood cells, 25,000 with 90 per cent polymorphonuclear neutrophils of which 50 were nonfilament. Two days later a gastrostomy for feeding and cervical mediastinotomy were done. On February 19, two days postopertively, she died suddenly, apparently of a vascular accident.

Case XII. G. E., aged eight, was admitted November 14, 1935, for laryngeal stenosis of several months' duration. On October 12, 1936, following laryngeal dilatation temperature rose to 102°F. In the next three days it rose in septic fashion to 104°F., and on October 16, 1936, *x*-ray revealed widening of the retrotracheal space with presence of gas. Cervical mediastinotomy was attempted but no pus could be found and the wound was packed with vaseline gauze. A second attempt on October 23 drained about 60 cc. of thick gray fetid pus and gas. Temperature returned to 99°F. in five days and to normal in eleven days and she continued to complete convalescence.

Case XIII. A. S., aged forty-four, was admitted May 12, 1939, with a history that all her teeth had been extracted four days before. The following day she awakened with marked swelling of the jaw, fever and general aching. The next day there was swelling of the floor of the mouth and neck with dysphagia and the third day this was increased and she noted pain in the chest substernally and under the left scapula. Examination revealed temperature

102°F., herpes labialis, toxic appearance with foul drainage from tooth sockets and marked swelling of the floor of the mouth preventing observation of the pharynx. There was marked swelling of the left neck and the white blood cells numbered 13,900 with 80 per cent polymorphonuclear neutrophils of which 34 were nonfilament. Smears and cultures showed no spirochetes and many viridans streptococci. Two blood cultures were negative. In the following three days dyspnea appeared and became pronounced, there was one chill, and the patient became dilirious and cyanotic and suffered severe abdominal pain and distention. This picture progressed till her death on May 17. Postmortem examination revealed pus in the retrovisceral space from neck to diaphragm, pericarditis, and bilateral empyema, each space containing more than 100 cc. of gray green pus, cultures of which showed Streptococcus hemolyticus and Streptococcus viridans in aerobic and anerobic culture.

Case xiv. E. G., aged forty-nine, was admitted March 20, 1938. Nine days before while eating fish, she choked on a bone, since which time there has been sticking pain in the throat. In the past three days pain was severe and there was almost complete dysphagia. Examination showed temperature 99.8°F. with swelling of the mucous membrane of the posterior pharyngeal wall; Wassermann 3, white blood cells 7,700 with 70 per cent polymorphonuclear neutrophils of which 34 were nonfilament. An *x*-ray revealed retropharyngeal thickening of soft tissues and gas. On March 22, cervical mediastinotomy produced 60 cc. of thick foul pus from about the esophagus. Cultures showed viridans streptococcus and bacteroids. Her temperature reached normal in eight days and she was discharged healed on April 16, four weeks after admission.

Case xv. R. T., aged seven, was admitted April 5, 1940, with a history of sharp pain in the throat since he had swallowed a sharp object while eating jam two days before. At that time his physician induced vomiting without improvement, and the mother discovered a section of glass missing from the jam jar. Examination revealed temperature 102°F., the patient was unable to move his head or swallow without pain, and there was tenderness over the neck. X-ray revealed a triangular piece of glass and emphysema of the retrovisceral space, both cervical and thoracic. The following day cervical mediastinotomy was done releasing gas and foul pus, and although the glass fragment was palpated, it was dislodged and moved down the esophagus preventing removal. His temperature fell to normal in five days, April 11, and he passed the glass by rectum. Sulfapyridine was administered for one day but was discontinued because of hematuria. He was discharged April 26 with a healed wound as cured.

Case xvi. J. S., aged forty-six, was admitted September 2, 1937, with a history that ten days before, while eating crab meat, he had swallowed a piece of shell. This caused coughing and a cutting pain in the throat with pain in the chest which persisted to admission. On examination, temperature was 101°F., pulse 100, respiration 30, but no physical signs could be found in the neck or the thorax. Esophagoscopy revealed esophagitis with ulceration. The following day there was more pain and emphysema with tenderness in the left neck anteriorly. The next day white blood cells 19,300, with 75 per cent polymorphonuclear neutrophils of which 15 were nonfilament forms, and *x*-ray revealed retrovisceral thickening and gas. In the next seven days there was steady improvement and he was discharged for observation as an outpatient. On September 15, the throat became very sore and in a few hours he coughed up a large quantity of pus. He was readmitted the following day, September 16, with marked dysphagia and induration along the anterior border of the left sternomastoid. The following day cervical mediastinotomy was done releasing gas and foul pus under tension. The temperature dropped from 101 to 98°F. in two days and he was discharged on September 24 improved.

Case xvii. A. W., aged forty-two, was admitted May 13, 1937, for esophageal stenosis of unknown etiology. She was examined endoscopically and the next day her temperature rose to 102°F. The following day *x*-ray revealed retrotracheal thickening and gas, and barium penetrating the esophageal wall. However, temperature slowly subsided and there was general improvement except for *x*-ray evidence on May 19 of increased gas and swelling in the retrovisceral space with narrowing of the trachea from outside pressure on the right. On May 21, eight days after the onset a cervical mediastinotomy was done releasing fetid yellowish gray pus from the retrovisceral space. Her convalescence was good until June 2, her

twelve postoperative day. In the next twenty hours she had three rather severe hemorrhages requiring secondary operation on June 3. This showed a slough of the carotid artery which required ligation of same for control. She developed a hemiplegia from loss of cranial blood supply and died the following day. Postmortem examination revealed continued posterior mediastinitis, fibrinous pleurisy but no fluid on the left and softening of the right cerebral hemisphere.

Case xviii. K. C., aged seventy-five, was admitted January 27, 1939, with a history of swallowing a fish bone five days before. For two days there had been severe pain on swallowing which then decreased. For the past three days she had fever and pain in the right upper chest anteriorly. Examination showed temperature of 100.4°F. and tenderness in the neck anteriorly. There were 14,800 white blood cells of which 77 per cent were polymorphonuclear neutrophils and 30 of these nonfilament forms, and blood sugar of 270. On January 28, cervical mediastinotomy was done releasing about 60 cc. of odorous pus and gas from the retrovisceral space. Temperature receded to normal in ten days and she was discharged February 12, 1939, as cured.

Case xix. G. B., aged fifty-four, was admitted October 9, 1939, for dilatation of cardiospasm associated with hiatal hernia. On October 12, esophagoscopic dilatation was done and two days later there was pain, tenderness, swelling and emphysema in the left neck anteriorly. White blood cells, 5,800 of which 88 per cent were polymorphonuclear neutrophils and 28 of these nonfilament forms. The *x*-ray revealed peritracheal gas and phlegmon. The following morning, October 15, the patient was delirious and cervical mediastinotomy was done releasing 10 to 15 cc. of foul brownish pus from the retrovisceral space. Temperature dropped to normal in eleven days but drainage persisted due to esophageal fistula. The purulent character disappeared within two weeks and healing was complete in 2 months. (Fig. 4.)

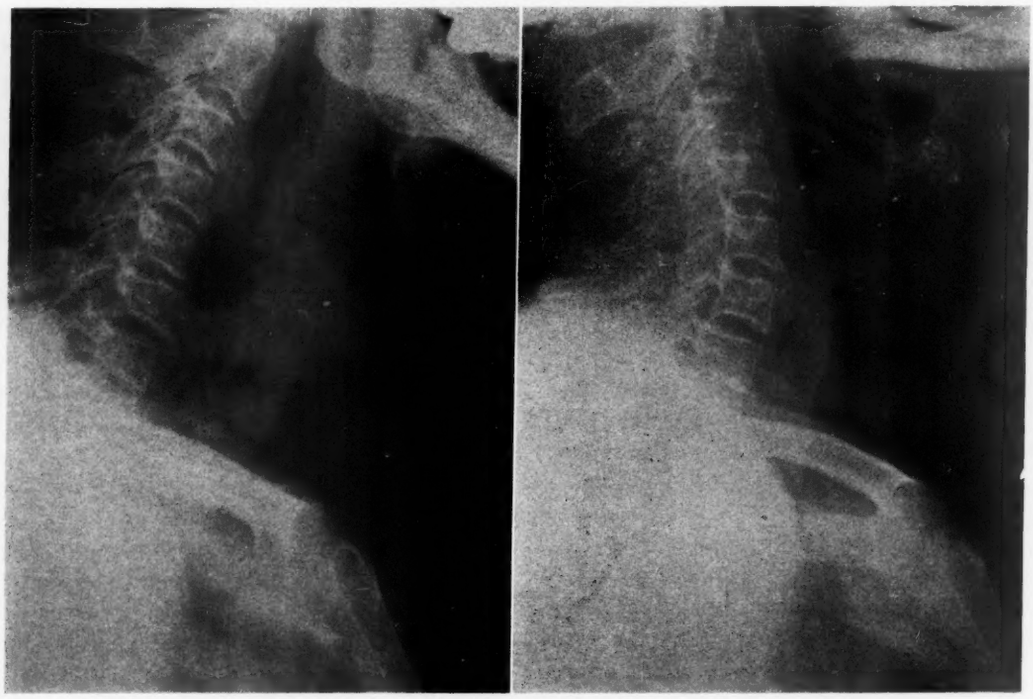

Fig. 4. Case xix. G. B., left, *x*-ray film of October 14, 1939, showing extensive retrovisceral thickening and gas. Right, *x*-ray film of February 2, 1940, showing decreased depth and gas.

Case xx. D. S., aged seventeen, was admitted May 14, 1937, with a history of swallowing a chicken bone a few hours before. Sticking pain had been present since. Temperature was 100°F. and there was slight tenderness in the neck. During esophagoscopy the bone passed on down the esophagus. Temperature increased to 102°F. and there was evidence by *x*-ray of gas in the mediastinum and left pleura and a white blood count of 17,000 with 85 per cent poly-

morphonuclear neutrophils of which 50 were nonfilament forms on May 15. On May 17, fluid was found in the left pleura and this was drained on May 18 by intercostal drainage revealing foul gray pus and gas. No mediastinotomy was done. The patient made slight temporary improvement followed by steady regression and finally hemorrhagic drainage through the thoracostomy ended in death on May 25.

Case xxi. K. M., aged thirty-eight, was admitted January 1, 1937, with a history that eleven days before while eating a veal cutlet a sharp object stuck in his throat. A physician and *x*-ray examination could find nothing abnormal. Six days later a lump appeared on the neck along the anterior border of the sternomastoid and two days later he began to expectorate foul pus. Additional *x*-ray revealed a piece of wire in the esophageal wall, and he was sent to the hospital. Examination revealed temperature 101°F., swelling on both sides of the neck more marked on the right which was tender and caused pain radiating to the right arm. Swallowing caused similar pain. The following day, twelve days after onset, cervical mediastinotomy revealed 15 cc. of fetid gray pus and a piece of wire. On January 13, temperature was normal, there was very little drainage, and the patient was symptom free. He was discharged to the care of his family physician. (Fig. 5.)

Case xxii. S. D., aged four was admitted April 1, 1938, with history of swallowing catsup bottle top seven days before. There was choking and cyanosis for a short time followed by vomiting and anorexia. Removal was attempted before admission following which there was cough and cyanosis. Examination revealed temperature of 101°F. with some reddening in the pharynx but no swelling or emphysema of the neck. There were a few rales over the right lower lobe area. White blood cells 18,400 with 42 per cent polymorphonuclear neutrophils of which 21 were nonfilament Four days later *x*-ray showed foreign body had moved into the stomach. There was a marked increase in temperature to 104°F. and in the white blood cells to 90,000 with 56 per cent polymorphonuclear neutrophils of which 45 were nonfilament. Two days later she was extremely toxic. There was no emphysema of the neck and *x*-ray showed widening of the mediastinum and the presence of gas. In this critical condition cervical mediastinotomy was done and thick cloudy fluid with considerable cellulitis and edema was revealed which showed a culture of hemolytic streptococcus. She died the same day, April 6, 1938.

Case xxiii. J. T., aged fifty-seven, was admitted July 12, 1937, for a carcinoma of the esophagus producing symptoms over the past six months. Following biopsy on July 12, the patient slowly developed temperature to 101°F. over the next three days with subcutaneous emphysema of the neck and *x*-ray evidence of retrotracheal widening and gas. There was also an increase of the lung marking on the left as compared to previous films. Two days later, July 17, there was increase in symptoms and local evidence and a cervical mediastinotomy was productive of foul pus and gas. Following this the patient improved rapidly over a period of two weeks and was discharged with continued drainage and without *x*-ray check-up. He was readmitted on September 10, 1937, seven weeks after mediastinotomy with a healed mediastinotomy and increasing dysphagia which was ascribed to his carcinoma. On September 11, a gastrostomy was done. On September 15, the patient had circulatory collapse with pain in the chest and left lower abdominal quadrant and physical signs suggesting atelectasis at the left base. *X*-ray revealed evidence of atelectasis or pneumonitis left base. The patient became progressively worse and died on September 25, having been moribund for five days. Postmortem examination revealed 400 cc. of thick foul pus in the posterior mediastinal space, purulent pericarditis and hemorrhagic effusion in both pleura, 800 cc. on one side and 1,800 on the other.

Case xxiv. H. B., aged eighteen months, was admitted December 28, 1938, two months after she had swallowed lye, with increasing difficulty in swallowing since. Her temperature was 101°F., physical examination negative and white blood cells, 16,500 with 40 per cent polymorphonuclear neutrophils of which 14 were nonfilament. On December 30, esophagoscopy showed ulceration and swelling of the mucosa. Immediately after the patient began vomiting and within four hours there was fever of 103°F., cyanosis, and labored breathing. This progressed rapidly, there was emphysema of neck and *x*-ray evidence of gas in the mediastinum, pericardium, and left pleura. Aspiration removed 40 cc. of milky fluid and gas from the

left pleura and similar material from the pericardium. Cultures of these showed hemolytic staphylococcus aureus and non-hemolytic streptococcus on both aerobic and anaerobic culture. The child was too ill to consider the multiple surgical procedures necessary for drainage and died in the next seven hours, December 31, 1938. (Fig. 6.)

FIG. 5. *a*, Case XXI. K. M., *x*-ray films of January 2, 1937, showing thickening of the retrovisceral space extending to the fifth rib posteriorly with gas and wire. *b*, *x*-ray films of January 12, 1937, showing decreased depth and gas and three wire sutures.

CASE XXV. C. VanF., aged twenty-five, was admitted November 3, 1939, with a history of sore throat and swelling anterior to the left mastoid beginning eight days before. He had pain in the left ear which had been host to otitis media at the age of three and again at the age of eight. His temperature was 99 to 100°F., pulse 74 and respiration 20. The pharyngeal wall, uvula, and left tonsillar fossa were red and swollen. There was poor dental hygiene and neck was hot, tender and indurated in the left anterior portion from the mandible to the base of the neck. The following day, November 4, a quantity of foul smelling pus was evacuated by superficial incision and apparently the character of the abscess was not recognized. Culture

showed Streptococcus viridans on aerobic and hemolytic streptococcus and bacteroids on anaerobic culture. Profuse drainage continued for three days then decreased, and he was discharged six days later as improved.

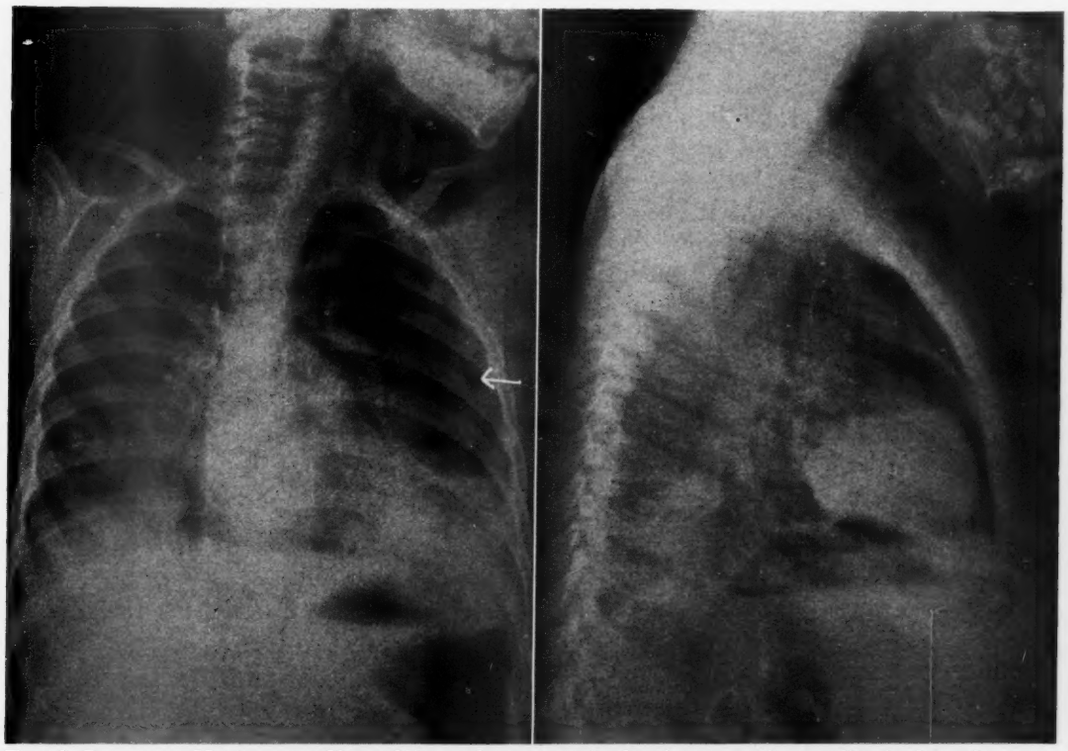

Fig. 6. Case xxiv. H. B., *x*-ray films of December 30, 1938, showing extensive mediastinal emphysema extending from skull to diaphragm, left hydropneumothorax and hydropneumopericardium.

He was readmitted December 1, three weeks later, stating that for one week after discharge he improved but was constantly hoarse. One week ago the slight pain on swallowing which he had suffered increased as did the hoarseness. One half hour before admission severe cough, dyspnea and some hemoptysis had occurred, and he was rushed to the hospital by his physician. Examination showed marked dyspnea and cyanosis and continued drainage from the cervical wound. Mirror laryngoscopy revealed fixation in adduction of both vocal cords with displacement of the larynx to the right. Emergency tracheotomy was done with marked relief. On December 2, *x*-ray revealed gas and phlegmon of the retrovisceral and visceral spaces—not communicating with tracheotomy wound. Displacement of the trachea to the right was also demonstrated. There were 10,800 white blood cells with 65 per cent polymorphonuclear neutrophils, 13 of which were nonfilament. Culture from the drainage revealed hemolytic staphylococcus aureus. His temperature returned to normal in five days and his progress was good. On December 13, opaque material was introduced through the cervical wound and *x*-ray taken outlining the retrovisceral space from the clavicle to the base of the skull. He was again discharged on December 22, with continued cervical drainage so that he might spend the Christmas holidays at home. He was readmitted on January 4 in the same condition. Because of persisting drainage the cervical wound was enlarged on January 17, 1940, and he was discharged on February 5 with lessened drainage continued paralysis of the cords and swelling of the epiglottic and subglottic regions. Treatment was continued over the next year for laryngeal stenosis requiring wearing of the tracheotomy tube. *X*-ray of August 30, 1940 showed persistence of soft tissue swelling about the larynx, chiefly to the right.

Case xxvi. H. S., aged twenty-three, was admitted June 5, 1937, with a history of extraction of the first left lower molar four days ago. Two days later there was swelling and pain in the left submaxillary area which has progressed to trismus and swelling under the tongue.

Examination revealed pus exuding from the swollen socket, trismus and tenderness with induration in the left submaxillary region extending in lessened degree to the left clavicle. There was also marked lymphadenopathy on this side, temperature 101°F., pulse 110, respiration 22. Culture showed Streptococcus viridans. He was given sulfanilamide 90 gr. daily, blood transfusions, and ice bag to the affected area, following which there was rapid and marked improvement so that the surgical drainage planned for the following day was postponed. Temperature reached normal in three days and he was discharged June 11 as cured.

Case. xxvii. R. DiG., aged forty-six, was admitted April 17, 1940, seven days after toothache began in the right lower third molar. The following day there was swelling about the mandible which increased, and two days later chills and fever occurred. The next day the tooth was extracted and much pus evacuated. However, the swelling increased, fever persisted and respiratory difficulty occurred just before admission. Examination showed swelling of the lateral and posterior pharyngeal wall, the floor of the mouth, and the entire neck anteriorly. The appearance of the gums suggested Vincent's angina and there was very poor dental hygiene. The patient was cyanotic and in a state of vascular collapse. The temperature was 101°F., pulse 140, respiration 50, white blood cells 13,450 with 89 per cent polymorphonuclear neutrophils of which 61 were nonfilament forms. *X*-ray revealed retropharyngeal and posterior mediastinal induration, and possible bronchopneumonia of the left lower lobe. He died within a few hours before anything could be done except the administration of sulfanilamide for two or three doses. Postmortem examination revealed Ludwig's angina, lateral pharyngeal space and retropharyngeal abscess extending into the posterior mediastinum, pericarditis and bilateral empyema. Blood cultures were overgrown with contaminants.

Case xxviii. L. S., aged forty-seven, was admitted January 13, 1937, with a history that four days before a chicken bone had stuck in his throat causing temporary choking. He made an effort to remove it with his finger but the symptoms persisted. That night there was dyspnea on lying down and at 4 A.M. his doctor tried to force the bone down the esophagus. Two days before admission *x*-ray revealed the bone and an attempt at esophagoscopical removal was unsuccessful. Since that time the patient was unable to swallow and there was pain in the throat and chest for which he was referred to the hospital. Examination revealed temperature 98.4°F., pulse 84, and respiration 22 with no swelling or tenderness of the neck but a swollen posterior pharyngeal wall. *X*-ray revealed retrovisceral phlegmon and gas and an opaque area suggestive of bone. Barium penetrated the posterior pharyngeal wall. He improved and was discharged on January 16 to his family physician. One month later he coughed up a portion of tooth-pick measuring two and one-half inches with complete relief except for occasional choking sensation over the past five months.

CONCLUSIONS

Acute mediastinitis is a fairly common disease as indicated by published reports and thirty-eight cases at Temple University Hospital in five years.

Knowledge of the practical anatomy of the fascial planes of the neck and thorax as reviewed is of vital importance.

Analysis of the etiology, symptoms, and results of our cases compared with 197 cases collected from the literature indicates that the mortality of undrained cases ranges between 60 and 80 per cent and that for the group in which adequate drainage was established varies between 33⅓ and 40 per cent.

Accurate diagnosis is possible if the lesion is considered in potential cases. *X*-ray study has been the most dependable method of investigation.

Immediate drainage by cervical mediastinotomy with the additional supportive measures outlined offers the best prospect for cure.

The author is very grateful to the following for their permission to analyze some of their cases: Doctors G. Mason Astley, W. Wayne Babcock, J. Norman Coombs, Matthew S. Ersner, Temple Fay, Chevalier L. Jackson, Savere F. Madonna, Robert F. Ridpath and William A. Steel.

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A GOITER fixed by capsular infiltration which makes the technic difficult is more liable to lead to disaster than those in which the gland is freely movable. Therefore if the gland is fixed, one had better wait until it loosens.

LOGICAL APPROACH TO SUBPHRENIC ABSCESS*

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A DISCUSSION that deals with thoracic surgery connotes a consideration of pathology above the diaphragm, but it is obvious when we consider the exact location of this structure that the region immediately below it is likewise well within the boundaries of the thoracic cage. For this reason the subject of subphrenic abscess is included in this volume, and the following presentation based on 124 consecutive operative cases covering the period from 1900 to 1941, from the Massachusetts General and associated Baker Memorial Hospitals is presented.

Objection might be raised that a study extending back to such an early period includes certain cases that were of necessity handled without the aid of modern surgical adjuncts. In an attempt to present the logical approach to the treatment of this condition, however, the inclusion of these earlier cases would seem helpful in affording a background for the evolution of present beliefs. Furthermore, with the single exception of the advances made in *x*-ray technic, there have been no outstanding surgical contributions that alter the fundamental considerations that this problem presented in this series.

Any study at a single institution over a lesser period would be productive of so relatively few cases that the statistical value of the data would tend to be limited and misleading. The cases involving those spaces beneath the liver edge that have come by common acceptance to be included under the term "subphrenic abscess" are likewise included; for although, strictly speaking, they lie below the thorax, they are so intimately associated both from an anatomic and a therapeutic point of view with the areas above the liver that they cannot well be dissociated from them.

It may seem trite to state that the most desirable surgical procedure in the treatment of an abscess which has been correctly diagnosed is that which promptly gives the most effective dependent drainage through the shortest approach consistent with the safety of the intervening structures. Certainly these general principles present no problem if we consider such a simple example as that presented by an axillary abscess. In the case of a subphrenic space involvement, however, the various factors mentioned are more complex and for that reason will be considered separately in greater detail.

STATUS OF INFECTION

In the case of a superficial infection, the stage of its development can be readily evaluated by palpation and the presence of fluctuation detected. However, with a subdiaphragmatic involvement, because of the resistance of the overlying ribs, the process does not permit such evaluation, and the question as to whether such infection has progressed to suppuration is often a difficult one to answer. It is well recognized that if frank pus in the subphrenic region is not drained surgically or does not rupture spontaneously into some favorable avenue of escape, the mortality will approximate 100 per cent. It does not necessarily follow that because there is inflammation beneath the diaphragm it will progress to abscess formation; in fact, Ochsner and Graves[1] state that over two-thirds of such infections subside without suppuration, and in this present series, one-fifth (20 per cent) of the cases with clinical signs of such infection followed a similar course. (Table 1.) Undoubtedly, many instances in which such a process was suspected but of necessity lacked con-

* From the Surgical Services of the Massachusetts General Hospital.

firmation failed to have such a diagnosis entered on their records, and thus the proportion of such cases errs in being too low in this present series.

Table 1
CASES DIAGNOSED AS SUBPHRENIC ABSCESS OR SUBPHRENIC INFLAMMATION

Classification of Case	No. of Cases
Surgical drainage of subphrenic abscess	124
No operation (subphrenic inflammation)	32
No operation for subdiaphragmatic abscess (lesion discovered at autopsy)	23
Operation but no pus encountered	6
Spontaneously drained via previous operative sinus	4
Total	189

In twenty-three cases in which no drainage of the subphrenic area had been instituted, autopsy examination revealed in one of the subdiaphragmatic spaces a collection of pus which in most instances was small in amount and of incidental importance as a cause of death.

Doubt is bound to exist in certain cases on the question of the actual presence or absence of pus, and in 6 instances of this series, the subphrenic region was explored and no abscess encountered. An occasional such properly executed exploration that reveals only "subphrenic inflammation" is infinitely more desirable than a timid attitude of vacillation that allows the process to persist unduly long or extend beyond its natural confines.

Spontaneous drainage occurred in four patients along the course of an apparently healed sinus tract that had resulted from the institution of drainage at the time of the original laparotomy.

ANATOMY

It is not surprising that considerable ambiguity should exist in the minds of many surgeons as to the exact limits of the constant subdivisions of the space beneath the diaphragm, for they are called upon relatively infrequently to explore certain areas of this region. In this series, for example, fifty-five surgeons were responsible for the treatment of the 124 patients and the greatest number drained by any one individual was only ten. The prime requisite, however, for prompt recognition and intelligent treatment of a subdiaphragmatic abscess is a thorough familiarity with the anatomy involved; for a diagnosis which localizes the infection beneath the diaphragm yet fails to specify which of the spaces it involves is bound to be attended with hesitancy in proceeding with surgery and relative ineffectiveness when it is finally adopted.

Martinet[2] and Barnard[3] at the start of the century presented such effective anatomical descriptions of this area that these have subsequently served as a basis for defining the limits of what is now understood to be included under the term "subphrenic region." Beneath the diaphragm the falciform suspensory ligament divides the area superior to the liver into two essentially equal right and left portions. (Fig. 1.)[4] On the left, there is but a single superior space as the triangular ligament of the liver does not encroach upon its surface but is attached to the posterior margin of the left lobe. On the right, the falciform ligament and its lateral extension divides the area into a right posterior superior space, and a retroperitoneal area within the laterally converging leaves of the ligament itself. (Fig. 2.)[4] Special note should be made that the anterior space is far larger than the posterior by virtue of the fact that the anterior portion of the triangular ligament lies well posterior to the dome of the liver, and that the posterior portion of the ligament by its dorsal obliquity restricts the right posterior space to a distinctly lateral position. (Fig. 3.) A retroperitoneal area that may become distended with pus exists between the diaphragm and diaphragmatic peritoneum; but this is a potential rather than a truly anatomic space, and infection in this region played a relatively minor rôle as this space was involved in but 3 per cent of these cases.

Beneath the liver on the right is a single right inferior space, less well demarcated

than those already referred to, yet none the less definitely bounded by the liver above, the round ligament and the ligament of

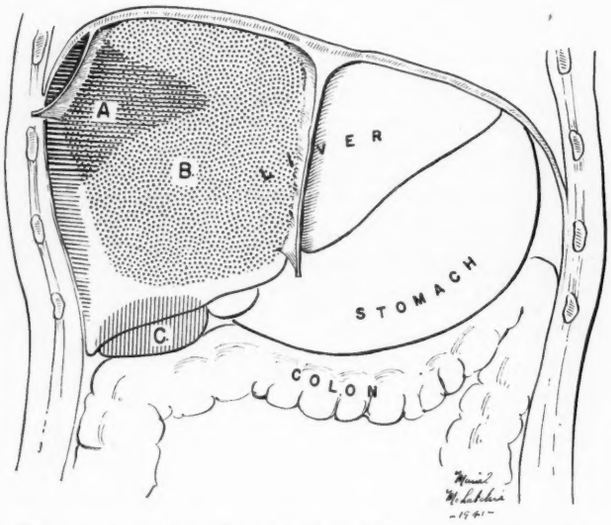

FIG. 1. Anterior view of the abdomen showing the three most commonly involved subphrenic spaces. A, right posterosuperior; B, right anterosuperior; C, right inferior. Actually the shaded portions of A and C lie behind the substance of the liver. (Faxon.[4])

the ductus venosus medially, the transverse colon below and the kidney posteriorly. On the left of the midline are two inferior spaces, the lesser peritoneal cavity which constitutes the left inferior posterior space, and the region anterior to this known as the left anterior inferior space with the liver above, the stomach below and the gastrohepatic omentum posteriorly.

DIAGNOSIS

Although certain reports would suggest that detection of a subphrenic abscess is easily accomplished, the writer's personal review of the records of these cases emphasizes the fact that in numerous instances great uncertainty existed in the mind of the surgeon as to whether infection was indeed present in the region of the diaphragm, and if so, whether it was above or below that structure. Even when he had satisfied himself that pus probably existed in one of the subphrenic spaces, he frequently was at a loss as to which of these was involved.

The age and sex of the individual were of no material diagnostic aid, the average age of thirty-eight years in this series and a preponderance of males (62 per cent) being merely a reflection of the group in which

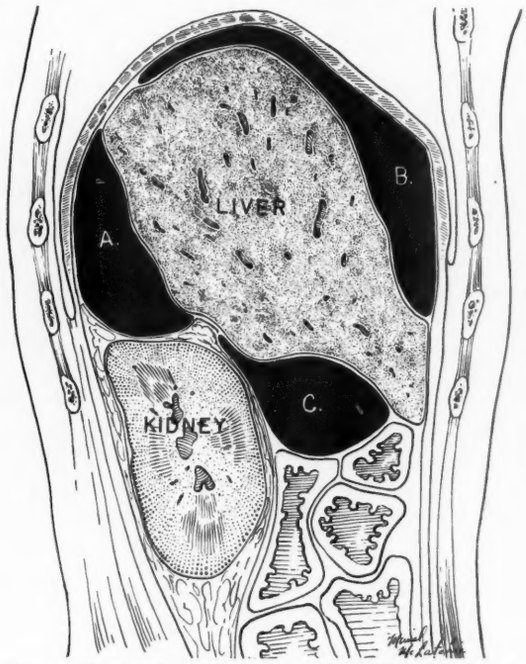

FIG. 2. Sagittal view of the right abdomen showing the three most commonly involved subphrenic spaces. A, right posterosuperior; B, right anterosuperior; C, right inferior. (Faxon.[4])

the various inciting causes of such abscesses are most prevalent.

Consideration of the diagnosis should be entertained in cases with unexplained clinical findings of localized infection in whom the presence of an antecedent intraperitoneal infection has been established or initiated by previous surgery, or in those unoperated cases in which the occurrence of such intraperitoneal sepsis has been strongly suspected. In one third (33 per cent) of the cases of this series, no previous surgery had been carried out, and the etiology was in some doubt in all save one-quarter (27 per cent) of these in which the history was perfectly characteristic of some given pathology.

In this series, appendiceal peritonitis most commonly furnished the primary source (31 per cent), with lesions of the stomach and duodenum second and of almost equal importance (30 per cent), and those of the liver and bile passages third (19 per cent). (Table II.) In the case of the

latter two sources, their close proximity to the subdiaphragmatic region makes the direct extension of infection to that area lymphatics or blood stream, there was no instance in this series in which the implantation of sepsis by either of these

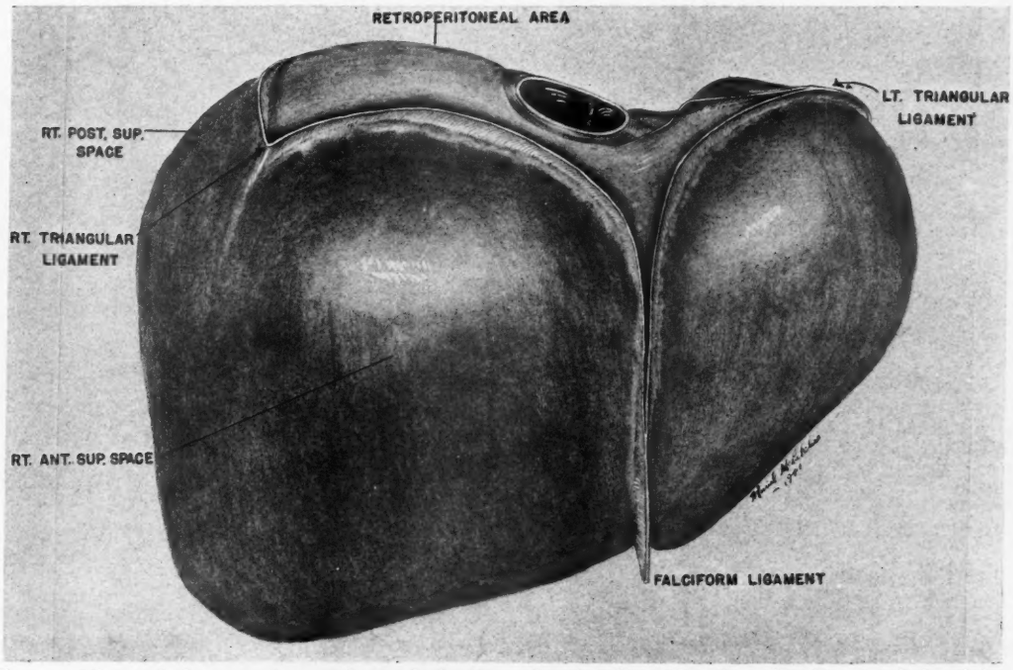

Fig. 3. View of the superior surface of the liver showing how the diverging leaves of the right triangular ligament limit the size and position of the most commonly involved posterosuperior space.

easily understandable. When appendicitis has been the inciting cause, the spread of infection from the distinctly more remote right gutter is best explained by the aspirating influence of respiration in the upper abdomen demonstrated by Overholt,[5] and the part the ventral curvature of the lumbar spine, with the patient in the supine position, plays in influencing the gravitation of material toward the subdiaphragmatic region.

Table II
ETIOLOGY OF SUBPHRENIC ABSCESS

	Cases	Percentage
Appendix	38	31
Stomach and duodenum	37	30
Liver and bile passages	24	19
All other causes	25	20

Although infection may find its way to one of the subphrenic spaces by way of the routes could be clearly established. In the cases in which an empyema was found in association with the subdiaphragmatic abscess, it seemed in all save possibly two cases to have been a result rather than a primary cause of the subphrenic abscess.

Granted a case in which a subphrenic abscess is suspected, the likelihood of its being present is strengthened by the finding of certain or all of the following factors: a high fixed diaphragm, fluid or gas beneath that structure, tenderness over a suspected space, and pleural effusion.

The position and motion of the diaphragm can be approximately determined by clinical examination of the chest, but fluoroscopic studies give more exact information on these points and in addition establish the presence or absence of fluid or gas in the upper abdomen. When the patient is too ill to permit such an examination, portable roentgenograms should be taken in both the anterio-posterior and lateral views with the patient's trunk sup-

ported in an upright position. *X*-ray facilities during the early part of this series were much more limited than in recent years, which explains the fact that in twenty-eight cases no roentgenological studies were made. In five cases, a sinus tract which had persisted following previous operative drainage was effectively utilized for the injection of a radio-opaque material to demonstrate the confines of the abscess cavity. (Table III.)

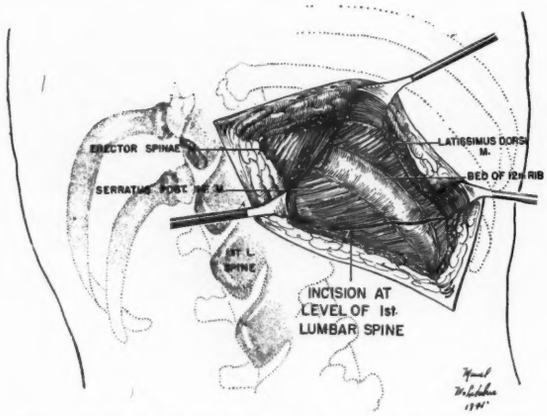

Fig. 4. Drawing of the initial steps in the retroperitoneal type of operation for a right posterior or inferior space infection. Note that the deeper dissection is at a level of the first lumbar spine rather than along the bed of the twelfth rib.

Table III
X-ray data in ninety-six cases* of subphrenic abscess

	Cases	Percentage
Diaphragm elevated	75	78
Diaphragm not elevated	4	4
Diaphragm not visualized	17	18
Fluid level and gas	28†	29
Opaque injection into sinus	5	5

* In twenty-eight cases no preoperative *x*-ray films were taken.

† One of these cases showed no fluid level, although gas could be demonstrated.

In nearly one-fifth (18 per cent) of the ninety-six cases in which *x*-ray studies were made, the outline of the diaphragm was obscured. When the diaphragm could be visualized, however, it was found to be elevated in practically all (95 per cent) of the cases. The erroneous idea is frequently entertained that gas and a fluid level can usually be demonstrated in a subphrenic abscess, when, as a matter of fact, this was true in but slightly over one-quarter (29 per cent) of the cases in which roentgenograms were taken. Muller et al.,[6] have demonstrated that free air beneath the diaphragm can be visualized by *x*-ray as long as two weeks following laparotomy, so that the possibilities for its presence must be weighed in order to attribute the proper significance to the finding.

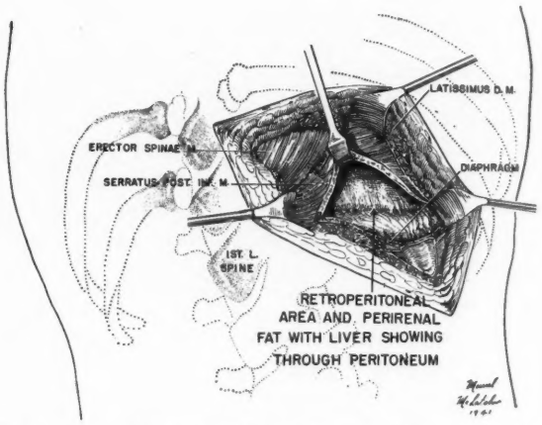
LATISSIMUS D. M.
ERECTOR SPINAE M.
SERRATUS POST. INF. M.
DIAPHRAGM
1ST. L. SPINE
RETROPERITONEAL AREA AND PERIRENAL FAT WITH LIVER SHOWING THROUGH PERITONEUM

Fig. 5. Drawing of a later stage in the retroperitoneal type of operation for a right posterior or inferior space infection. Note that the lower fibers of the diaphragm have been cut across but that the peritoneum has not been incised.

That the type of contamination was the most important factor in the occurrence of fluid and gas in the subphrenic abscess is brought out by the fact that nearly one-half (thirteen) of the cases with this finding followed a perforated gastric ulcer in which gas and fluid played a part at the time of the initial insult to the peritoneal cavity. There were, however, an equal number (thirteen) of subdiaphragmatic abscesses following perforation of a gastric or duodenal lesion in which no such finding was present. Neglect in instituting drainage could not be shown statistically to account for fluid and gas for the cases in which these were present were actually drained earlier following their initial infection (twenty-two days) than those in which they were absent (twenty-seven days).

As with circumscribed infections in other conditions, localized tenderness is of great diagnostic significance in a suspected case of subphrenic abscess. Inasmuch as with involvement of the superior spaces the thoracic cage intervenes as a barrier between the skin and the underlying infection, it is frequently easier to elicit tenderness by compression of the chest wall than by any lighter forms of palpation. Careful interpretation of the findings of such examination is helpful in further suggesting which of the spaces is involved, for with the abscess in the right posterior superior space, tenderness will be found over the twelfth rib posteriorly; while with infection in the anterior superior spaces, it will be brought out by firm pressure over the respective right and left anterior costal margins.

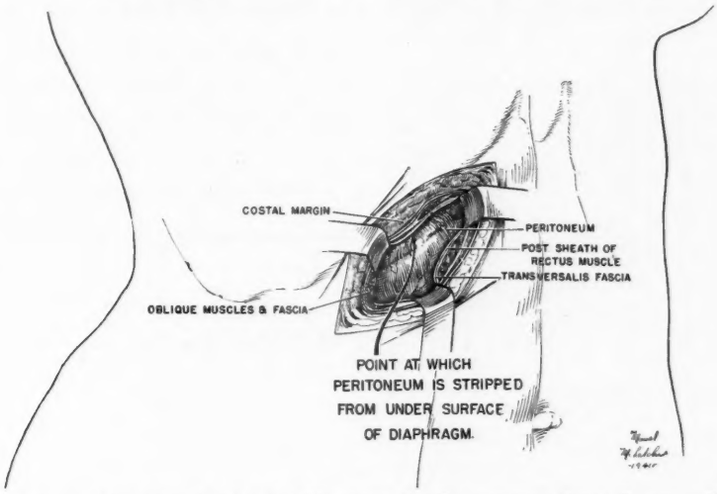
COSTAL MARGIN
PERITONEUM
POST SHEATH OF RECTUS MUSCLE
TRANSVERSALIS FASCIA
OBLIQUE MUSCLES & FASCIA
POINT AT WHICH PERITONEUM IS STRIPPED FROM UNDER SURFACE OF DIAPHRAGM.

Fig. 6. Drawing of the initial steps in the retroperitoneal type of operation for an anterior space infection. Note that the posterior fascial layers must be incised before the peritoneum can be stripped from the undersurface of the diaphragm.

The finding of pleural effusion may be more confusing than helpful in arriving at the proper diagnosis, for its presence may be mistakenly ascribed to a primary source of infection above the diaphragm. Opinions[1,7,8] differ as to the frequency with which fluid is found above the diaphragm in association with a subphrenic abscess. The data from this series of cases are inconclusive on this point, for in a definite number of the eighty-two cases in which there was no note of such fluid being actually demonstrated, it undoubtedly may have existed. In over two-thirds (72 per cent) of the cases, however, in which thoracentesis or visualization of the pleural cavity was carried out, some pleural effusion was apparently present and was usually (87 per cent) found in conjunction with an abscess involving one of the superior spaces. (Table iv.) That it did not of necessity occur in the late neglected cases is suggested by the fact that in the patients in which it was demonstrated, and who were operated upon for their original

Table iv

Findings as regards pleural effusion associated with subphrenic abscess

Clinical Data	Number of Cases	Percentage of Total Cases
No thoracentesis or operative visualization of pleural cavity	82	66
Empyema at time of subphrenic drainage	10*	8
No fluid by thoracentesis (4) or visualization (5)	9	7
Pleural effusion by thoracentesis (23) or visualization (0)	23†	19

* 70 per cent of these were grossly neglected cases occurring prior to 1924.

† 87 per cent of these involved one of the superior spaces.

pathological condition at the same hospital entry at which the abscess was drained, the elapsed time from the inciting cause to the drainage of the abscess (twenty-nine days) was essentially the same as in those in which there had been no demonstrable evidence of any pleural effusion (thirty days). These intervals in this series of cases between the time of contamination of the subphrenic region and the institution of drainage have remained disturbingly constant over the years and are distressingly long, for they reflect delay and difficulties in diagnosis rather than the actual time that it takes for such an abscess to become established.

The practice of thoracentesis in a case in which pleural effusion is suspected by clinical examination or *x*-ray studies is an altogether justifiable procedure to differentiate between an empyema and a simple effusion. If this procedure is adopted, special care should be exercised to avoid penetrating the diaphragm in a further search for an abscess beneath that structure, for if such is encountered and the then contaminated needle be withdrawn through an uninvolved serous cavity, the added complication of empyema or peritonitis will frequently result. Preoperative exploratory aspiration of the subphrenic spaces for this reason is never justifiable, but if employed in the face of this objection it contributes information only if pus is actually secured, for it cannot be assumed because of a failure to obtain pus that no abscess is present. It is of some suggestive interest that the mortality rate (66 per cent) was approximately twice as great in the twelve cases in which it was practiced preoperatively as it was both in the cases in which it was not adopted (33 per cent) and in those in which aspiration was done with relative safety under direct vision at the time of operation (36 per cent). From a study of the records, it is seen that this difference in mortality was in part due to the complications attendent on such ill advised, blind explorations, but it is only fair to add that nearly three-quarters (70 per cent) of the cases with an associated empyema present at the time the subphrenic abscess was drained occurred prior to 1924, and that seven of such empyemas were due to neglect with rupture of the subphrenic abscess through the diaphragm rather than as a result of thoracentesis.

TABLE V
INFLUENCE OF ETIOLOGY ON SPACES INVOLVED IN SUBPHRENIC ABSCESS*

Etiologic Factor	Number of Spaces Involved†	Spaces Involved					
		Rt. Post. Sup.	Rt. Ant. Sup.	Rt. Inf.	Lt. Sup.	Lt. Ant. Inf.	Retroper.
		Per Cent					
Appendix as original infection	54	46	17	20	4	3	10
Stomach and duodenum	39	26	36	15	9	14	0
Liver and bile passages	29	24	48	18	3	7	0
All other causes	32	40	16	19	21	0	4

* A total of 154 spaces were involved in the 124 cases. There was but one case recorded as a "subphrenic abscess" involving the left posterior inferior space.

† Twenty-seven cases had abscesses in more than a single space.

In certain instances, all clinical diagnostic measures may fail to clarify in the surgeon's mind which of the subphrenic spaces is involved, and when such is the case, it is important to appreciate the fact that the original cause for the abdominal infection will materially influence the site at which the abscess is most frequently found. (Table v.) Thus when appendiceal peritonitis was the primary pathology, the subdiaphragmatic abscess involved the right posterior superior or right inferior space in two-thirds (66 per cent) of the instances; while with the liver and bile passages as a source, approximately one-half (48 per cent) of the secondary abscesses became established in the right anterior superior space rather than the more dependent but usually drained right inferior space. The central position in the upper abdomen of the antrum of the stomach and duodenum undoubtedly explains not only the approximate uniformity with which the different spaces were involved but also the relative high incidence (23 per cent) of

left-sided abscesses following gastric surgery or the perforation of an ulcer.

The right posterior superior space was the most frequent site of a subphrenic abscess (36 per cent) with the right anterior superior (27 per cent) and right inferior (18 per cent) next in relative importance. (Table VI.) In over one-fifth (22 per cent) of the patients, more than one space contained pus, either because of direct extension from one space to the adjacent one, or because of implantation of infection in more than one area at the time of the original spread of sepsis.

TABLE VI
INCIDENCE OF SPACES INVOLVED IN SUBPHRENIC ABSCESS*

Space	Cases	Per Cent
Right posterior superior	55	36
Right anterior superior	42	27
Right inferior	28	18
Left superior	15	9
Left anterior inferior	9	6
Left posterior inferior	1	1
Retroperitoneal	4	3

* A total of 154 spaces were involved in the 124 cases.

The right subphrenic spaces were involved more than five times as frequently (84 per cent) as those on the left (16 per cent), and because of the consistently lower incidence of left-sided involvement found in this series of cases as well as those reported in the literature,[9] there has been a tendency to minimize the importance of abscesses on this side. This tendency is especially unfortunate inasmuch as the anatomic subdivisions of the left subphrenic region are less sharply demarcated and are more difficult to approach without untoward contamination of uninvolved areas than on the right. The fact that the mortality in the twenty-two cases with left-sided lesions in this series (50 per cent) was one and one-half times as great as for those occurring on the right side alone (34 per cent) lends weight to the importance of careful consideration in the treatment of this smaller segregated group.

TREATMENT

In an attempt to satisfy the requisites of ideal treatment by prompt, effective, safe drainage, three general types of operation have been evolved. Each stresses with different emphasis the factors mentioned, and only by comparison of the theoretical advantages and actual results of each can their relative merits be determined.

The transperitoneal type of approach gains access to the infection by traversing the upper portion of the peritoneal cavity and entering directly into the abscess cavity. Although it does not necessitate the delay of a two-stage procedure and usually gives direct and dependent drainage, the contamination of the general abdominal cavity that frequently results is so obviously prejudicial to the successful outcome of the case that no further word of condemnation of this method when it can be avoided is required. It is true that infection in both the left inferior spaces can be drained only by some such type of approach, but it is to be hoped that in such cases adhesions will have formed between the inflammatory area and the anterior abdominal wall that will permit access to the pus without contamination of the uninvolved portion of the peritoneum. In this series, most of the twenty-five cases that were drained transperitoneally were found either in the first half of the study when the advantages of other methods were less fully appreciated (ten cases), or else subsequent to those earlier years in patients with their infection to the left of the midline (seven cases).

The main difference of opinion as to the logical approach in the drainage of a subphrenic abscess centers about whether a transpleural or retroperitoneal type of operation should be adopted. The transpleural operation is carried out by resection of that portion of the ninth, tenth or eleventh rib that is immediately over the lower portion of the suspected abscess. When the underlying pleural cavity has been exposed, it may be safely traversed if it has become obliterated at this point by

an inflammatory reaction and the abscess entered by incising the diaphragm. It cannot be assumed, however, that such obliteration will necessarily take place as a secondary manifestation of the infection of the subdiaphragmatic region; and if doubt exists at the time of operation as to whether the exposed angle of the pleural cavity is obliterated, the transpleural operation should be carried out as a two-stage procedure. The first stage consists of the removal of a portion of the appropriate rib, the suturing of the pleura to the diaphragm about the edges of the wound and the insertion of a pack into the area. At the second stage, two to seven days following the first, the pack is removed, and the further incision into the abscess cavity carried out. Although less than one-third (30 per cent) of the forty-three cases of transpleural drainage of this series were done as a two-stage procedure, there is now general unanimity of opinion that the operation carries a lower mortality when done in two stages than as a single operation. A recognition of this fact has been marked by over a sevenfold increase in the use of the two-stage operation in cases drained by this type of approach during the last twenty years (44 per cent) as compared with the preceding period (6 per cent).

The chief argument advanced for the transpleural type of procedure is that it utilizes the most direct route to the imprisoned pus. It is contended that the interval between the stages, when done as a two-stage operation, is relatively unimportant if the diagnosis has been promptly made; but obviously in many instances the diagnosis has been long deferred and in these it is essential that the pus should be evacuated with the least possible delay once surgery has been adopted.

If the preoperative localization of the abscess has been incorrect, such an approach is ill suited for further exploration; and when a combination of spaces is involved, such an incision cannot drain effectively more than a limited portion of the infection present.

The chief danger in the transpleural approach, however, lies in the possibility, which will be discussed in greater detail later, of inadvertently contaminating an uninvolved pleural cavity and thus initiating an empyema.

The retroperitoneal, or extraserous type of operation, when done for drainage of the commonly involved right posterior or right inferior spaces, is carried out through a skin incision overlying the twelfth rib. It is important that preoperative roentgenograms should be studied to ascertain the length and position of this rib; for if it be a rudimentary structure, as was true in one case in this series, the eleventh may be mistaken for the twelfth rib and thus result in a false orientation by the surgeon in his approach to the region. The twelfth rib is removed subperiosteally in its entirety, and further dissection is then carried out in a horizontal direction at the level of the first lumbar vertebra, rather than along the bed of the resected rib. (Figs. 4 and 5.) The importance of the location of this deeper dissection lies in the fact that, as Melnikoff[10] has shown in a study of cadavers, the pleura on the right extends to below the twelfth rib in nearly two-thirds (62 per cent) of all cases but is never found as low as the level of the first lumbar vertebra, so that a properly placed incision at this lower level will always avoid inadvertent contamination of the pleural cavity. With division of the lower fibers of the diaphragm, the retroperitoneal area above the kidney is entered, and by blunt dissection the peritoneum is readily stripped from the under portion of the diaphragm up to the point where it no longer separates easily because of the inflammatory reaction occasioned by the infection. It is here that the peritoneum is broken through and the abscess cavity entered directly. When either the lateral aspect of the right anterior or the right inferior space is involved, the initial surgical approach is the same but the deeper exploration of the area by blunt dissection is carried out more anteriorly in

the former instance and below the liver edge superior to the kidney in the latter.

If either the right anterior superior or the left superior spaces are involved, an incision is made parallel to and just below the anterior costal margin. (Fig. 6.) This extends down to but not through the peritoneum which is then dissected free from the undersurface of the diaphragm in a manner similar to that already described for the posterior approach. It is important in securing the proper line of cleavage for this dissection that the posterior layer of the rectus sheath and the transversalis fascia directly overlying the peritoneum both be incised lest the separation of the peritoneum from the diaphragm be thwarted by the attachment of these fascial layers.

The advantages of the retroperitoneal type of approach lie in the fact that the operation is a single-stage procedure, thus avoiding any unnecessary delay in evacuating the pus once the diagnosis has been made. Furthermore, entrance into the abscess cavity is effected at its most dependent portion, which is of more fundamental importance in the effectiveness of the resulting drainage than the directness of the route by which the infection is attacked.

The greatest advantage of the retroperitoneal approach, however, lies in the fact that it carries a lower risk of contaminating uninvolved serous cavities than either of the types already discussed. In the fifty-six instances in which the extraserous operation was employed, the pleural cavity was inadvertently exposed to contamination in less than one-tenth (7 per cent) of the cases. When such contamination did occur, it was either because of faulty technic in the removal of the rib from its enveloping periosteum, or in the unfortunate choice of the bed of the rib rather than the level of the first lumbar vertebra as a site for the deeper dissection. This is in contrast to similar encroachment on the pleural cavity in over one-third (35 per cent) of the transpleural operations, regardless of whether they were done in one or two stages. No instance of contamination of the pleural cavity at the time of operation took place in the transperitoneal type of operation, but this procedure carried a prohibitively high incidence of peritoneal soiling (80 per cent). (Table VII.)

TABLE VII
INCIDENCE OF CONTAMINATION OF UNINVOLVED SEROUS CAVITIES ACCORDING TO TYPE OF OPERATION

Site of Contamination	Transperitoneal Per Cent	Transpleural Per Cent	Retroperitoneal Per Cent
Both pleural and peritoneal cavities	0	2	0
Pleural cavity alone	0	35	7
Peritoneal cavity alone	80	0	2
Neither cavity	20	63	91

That this repeated emphasis on the importance of avoiding contamination of an uninvolved serous cavity is of paramount importance is brought out by the fact that the mortality was over two and one-half times as high in cases in which it occurred (63 per cent) as in those in which it was avoided (24 per cent). (Table VIII.)

TABLE VIII
EFFECT OF CONTAMINATION OF UNINVOLVED SEROUS CAVITIES AT TIME OF DRAINAGE OPERATION ON MORTALITY RATE

Site of Contamination	Cases	Mortality Per Cent
Both pleural and peritoneal cavities	1	100
Pleural cavity alone	19	63
Peritoneal cavity alone	21	62
Neither cavity	83	24

The valid objection might be raised that certain factors modify the accuracy of these arbitrary figures concerning contamination of uninvolved serous cavities. One such factor lies in the interpretation of the frequently employed term "contamination," for it was often difficult from the records to

decide whether a serous cavity which had been inadvertently opened and promptly closed had, in fact, been subjected to any contamination by the infectious process the operation primarily sought.

A second source of potential error is found in the tabulation of those nine cases in which more than one type of operation was carried out because of involvement of multiple spaces that could not be approached surgically through a single incision. To permit a relative comparison between the incidence of contamination with each type of operation, every case was classified under a single operative heading which departed from the original surgical approach only if this had been directed toward draining a space that proved subsequently to be of lesser importance than that presented by a second abscess, or if it had been unsuccessful in reaching the abscess cavity. In this latter connection it appears that the medial portion of the right anterior superior and left superior spaces that lie posterior to the convex surface of the liver are probably best approached by the transpleural operation, for in four such cases in which the retroperitoneal operation was adopted, it proved ineffective and was followed by satisfactory drainage from above the diaphragm.

Subsequent to 1917, when an affiliated convalescent home which made possible the early transfer of postoperative patients was discontinued, the average hospital stay for the surviving cases following drainage of a subphrenic abscess by the retroperitoneal route was forty-two days as compared with fifty-seven days for those in which the transpleural operation was adopted. There has been a definite trend in the more recent years of this series with both the retroperitoneal and transpleural type of operation toward a decrease in the stated average periods of hospitalization following drainage, and it is to be hoped that with early diagnosis and adequate surgery, the patient should have sufficiently recovered to permit him to be discharged from the hospital within three weeks following operation.

In comparing the different types of procedures, the rôle of anesthesia has applied uniformly regardless of the operation adopted. There has been in all cases an increasing trend from a general anesthetic to a greater use of local novocaine infiltration for at least the initial stages of each of the operations.

In each of the procedures, two or three cigarette wicks together with the occasional use of a piece of soft rubber tubing were inserted to maintain adequate drainage until the process had completely subsided.

There has been too small a number of cases (four) to draw any conclusions as to the efficacy of chemotherapy as an adjunct to surgery, but experience in dealing with the somewhat analogous situation found in appendiceal abscesses would lead one to suppose that the use of the sulphonamides might play an important part in the future in lowering the mortality of cases with collections of pus in the subphrenic region.

MORTALITY RATE

It is difficult to evaluate the relative merits of the different procedures in terms of their respective mortality rates, for in many instances the fatality was due primarily to the original pathology and death would probably have ensued even if no subphrenic abscess had developed. To segregate such patients, however, in an attempt to arrive at a corrected mortality rate would introduce too great an element of fallible evaluation to make the resulting figures reliable. In certain additional cases, complications other than those already discussed as having a peculiarly close relationship to the subphrenic abscess have played an important part in the ultimate outcome of the case, but here again a weighting of the figures would seem to be more misleading than corrective. For these reasons, all deaths in this series are included in computing the mortality rates of the different types of drainage of subphrenic abscesses, as it was believed that in a series of cases of this size, these ex-

traneous factors would tend to occur so relatively uniformly within the three groups that they would exert a comparable effect on each. Certainly it is true that in almost all the fatalities the presence of a subphrenic abscess in an already depleted patient was the deciding factor in the untoward outcome of the case.

Ochsner[1] has shown that the mortality rate can be reduced to below 20 per cent, and the figure of 24 per cent for the two-thirds (67 per cent) of the cases in this series in which no contamination of a serous cavity took place is further evidence that regardless of other considerations, with proper surgical treatment, not over one out of four patients who develop a subphrenic abscess should die. The mortality of 37.1 per cent for this entire series is unduly high because of the difficulties in diagnosis and surgical management of these patients in the period from 1900 to 1920, and the unfortunate number of instances in which contamination of uninvolved areas has continued to take place since that time.

A growing conviction borne out by available statistics that in all save exceptional instances the retroperitoneal type of approach provides the safest method of draining a subphrenic abscess has led to an almost unbroken increase in the frequency with which this method has been employed in these cases. (Chart 1). The conclusion that the consistent improvement in the mortality rate can be attributed solely to this factor is, of course, unjustified, for the better understanding of postoperative care during more recent years might be cited as but one of a number of additional factors that have tended to decrease the death rate. The fact remains, however, that the greater avoidance of a spread of infection with the extraserous approach is the chief reason for the smaller proportion of fatalities during the more recent years.

Even at the hospital from which these figures and beliefs have emanated, there still exists some difference of opinion as to the wisdom of accepting the retroperitoneal type of approach in all cases in which it might be employed. During the past six

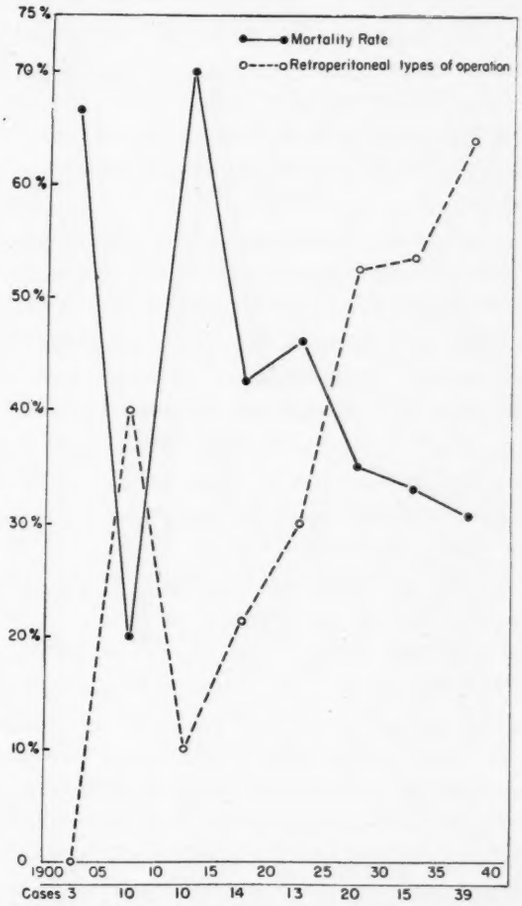

Chart 1. Showing the increasing incidence in the use of the retroperitoneal type of operation and the decreasing mortality rate over the period studied.

years, there were over one-third (35 per cent) of the cases of this series that were treated by some other form of operation, and it remains to be seen whether with further time and added evidence the extraserous procedure will completely supplant the transpleural and transperitoneal types of drainage in all save the small groups of cases already referred to for which it does not seem applicable.

SUMMARY AND CONCLUSION

The statistical data compiled from 124 consecutive cases of subphrenic abscess operated upon at the Massachusetts General Hospital from 1900 to 1941 are pre-

sented to illustrate the salient features of diagnosis and treatment of this condition. From these data the following conclusions can be drawn:

1. At least one-fifth of all cases with "subphrenic inflammation" subsided without suppuration.

2. Surgical drainage is indicated as the accepted treatment for a subphrenic abscess.

3. A thorough knowledge of the location and anatomic boundaries of the six spaces into which the subdiaphragmatic area is divided is essential for prompt diagnosis and early intelligently planned surgical drainage of a subphrenic abscess.

4. Inadvertent contamination of an uninvolved serous cavity during the drainage of a subphrenic abscess more than doubles the mortality rate.

5. The retroperitoneal type of approach may be effectively employed in the drainage of over four-fifths of all subphrenic abscesses. It is the logical surgical approach to most cases of subphrenic abscess as it is attended with the lowest incidence of contamination of uninvolved serous cavities and carries with it a lower mortality rate than the other two types of operation.

6. Although the posteromedial portions of both the right anterior superior and left superior spaces can be reached by the retroperitoneal type of approach, the transpleural type of operation would seem more desirable in the drainage of these areas.

7. The transperitoneal type of operation, due to anatomic considerations, is the only one available for the drainage of the left inferior spaces. It should not be employed as a surgical approach to any of the other spaces.

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TUMORS OF THE THORACIC CAGE

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TUMORS of the chest wall may be divided into two broad groups: those affecting the soft tissues (exclusive of tumors of the breast) and those arising from the bony and cartilaginous framework. The former include all those affecting similar tissues elsewhere; the latter those affecting other bony and cartilaginous structures. In addition it should be remembered that growths originating within the chest may give rise occasionally to outward swellings either by extension into the wall or by expanding the wall in the region of the growth. A young woman who came under my care some years ago with what proved to be a lymphosarcoma of the mediastinum had an obvious bulging of the sternum which receded with radiotherapy, without any actual invasion of the tissues of the wall. In addition to these primary neoplasms it is necessary to consider in differential diagnosis the metastatic lesions which so frequently involve the ribs and sternum, as well as inflammatory swellings which may simulate new growths.

Soft Tissues. The various tumors of the skin found elsewhere occur on the chest. They include papillomas, occasional basal cell and epidermoid carcinomas, benign and malignant melanomas. The problems of diagnosis and treatment present no unusual features and need not be considered here.

Lipomas occur fairly frequently and in general resemble other fatty tumors. The need for removal of these will depend chiefly upon their location and size but occasionally, in the case of neurolipomas, upon pain and tenderness. They sometimes attain a great size. A lipoma arising deeply in the intercostal space may take on a dumbbell or collar button form, part of the growth lying superficial and part deep to the ribs. It is obvious that removal of such tumors presents a special problem. Fibromas and fibrosarcomas occur rather infrequently. If the lesion is really a fibroma a very simple local removal is, of course, all that is necessary. The borderline type of growth in which there may be difficulty in arriving at a certain diagnosis of benignancy or malignancy may present a real problem as illustrated by the following two case histories:

1. Miss V. Z. No. 093799, was admitted to the hospital on September 17, 1934 with the history that fifteen months previously she had noticed a lump on the right chest near the inner border of the breast at about the level of the sixth costal cartilage. Five months later when it had attained the size of a lemon it had been removed locally. The pathological diagnosis was fibrosarcoma. Three months after this operation another lump was noticed at a slightly higher level. This had increased slowly in size. On examination there was a mass three inches long extending laterally from the sternum along the cartilage of the fourth rib and extending over the cartilage of the fifth rib. It was definitely attached to if not actually arising from cartilage. *X*-rays were negative. On October 6, 1934 a wide resection of the mass along with the cartilages of the fourth, fifth and sixth ribs and an attached block of soft tissues was carried out, the chest cavity being widely opened in the process. The patient made a good recovery. The specimen was composed of a dense mass of fibroblastic tissue arranged in whorls and infiltrating the adjacent soft tissue and cartilage. There were occasional mitotic figures. It was regarded as a low-grade fibrosarcoma. Radium packs were applied when healing was complete. On April 6, 1937 she was re-admitted with a hard, oval mass three and one-half by one inch apparently fixed to the cartilage of the third rib. On April 9, 1937 a wide resection of this mass and the cartilages of the second and third ribs was done. The pathological picture was the same as that

of the mass removed previously. There was no further evidence of recurrence until November, 1939, at which time the patient was lost track of.

2. Mr. Wm. R., No. 091437, age forty-six, was admitted on July 11, 1934 with the history that for eight years he had had a growth on the chest wall. Three years previously it had been removed. One and one-half years later it had begun to recur in the scar and had increased slowly to the size of a walnut. Examination revealed a linear scar four inches long, one inch below the left clavicle and parallel with it. In the center of this scar was a mass the size of a walnut. It was hard, nodular and freely movable on the deep structures. There was no enlargement of lymph-nodes. A fairly wide local removal was done including a portion of the fascia of the pectoralis major. Microscopically, the growth was a borderline type but was regarded as a low-grade fibrosarcoma. The patient was readmitted in June, 1936, and stated that six months after the last operation a recurrence had appeared again in the scar and had slowly increased in size. It was once more the size of a walnut. This time a very wide removal of skin and underlying pectoralis major muscle was done. The pathological picture was similar to the original and again gave the impression of very low-grade malignancy. Two courses of H. V. therapy were administered to the chest wall after the wound had healed. In October, 1940, there had been no recurrence.

These cases illustrate the tendency of this type of growth to recur after anything but the most complete extirpation. They are not radiosensitive and must be treated surgically. The cellular, more rapidly growing type of fibrosarcoma is more likely to respond to radiation.

Tumors of blood and lymph vessels of the chest wall apart from ordinary nevi would seem to be very rare. They present the usual features and if not too extensive should be removed. An infant that came under our care some years ago had an enormous hemolymphangiolipoma involving most of the chest wall and abdomen. It was present at birth and grew at least as rapidly as the child. An exploration done in the hope of removing it piece-meal showed that it invaded the underlying muscles and that removal was quite impossible. It was benign and the child was quite healthy although greatly deformed by the growth, presenting the appearance of being bloated.

Ribs and Cartilages. Primary tumors of ribs and cartilages are distinctly uncommon. They comprise all those tumors of bone and cartilage that occur elsewhere and in general present the same characteristics and tendencies.

Chondromas and osteochondromas would seem to be the commonest of the benign lesions. They may occur anywhere in the course of the rib but are more frequent over the anterior chest wall in the neighborhood of the costochondral junctions. The swelling produced is usually more or less fusiform in shape and painless unless pain be produced by pressure on adjacent structures. They are hard, somewhat nodular and not tender. They are likely to be discovered accidentally and are, therefore, of uncertain duration. The *x*-ray appearance presented depends upon whether they are composed exclusively of cartilage, of cartilage with calcification or of cartilage and bone. The pure cartilage growth may not be recognizable in the *x*-ray; the chondroma with calcification tends to throw a dense shadow of a somewhat flocculated character; the one in which bone has been formed gives a shadow of varying density but of more regular arrangement than the calcifying chondroma. These tumors are stated to have a tendency to undergo malignant changes but it is difficult to know how often this occurs. One seen recently had been present for twenty years with little change and was quite benign. On the other hand one seen just previously had been present for many years without any apparent change but had then commenced to grow rapidly and when seen was an enormous chondrosarcoma. However frequent this tendency may be these tumors should be removed when encountered. Because incomplete removal is likely to

be followed by recurrence, even in the case of benign growths, they should be removed along with the enclosing periosteum, attached muscles and underlying pleura, unless the latter strips away very readily, since an attempt to save an adherent pleura is likely to result in the leaving behind of tumor cells. They are not radiosensitive and radiotherapy has no place in their treatment.

Giant cell tumors of ribs are very uncommon. Only eleven cases have been reported. They may occur anywhere in the course of the rib and give rise to the well known expansile lesion. Attention is attracted to them by pain and not infrequently by pain that results from a pathological fracture. If the tumor is palpable it is likely to be elastic or may be elastic with the sensation of a yielding shell. The *x*-ray appearances are such that one cannot distinguish these from other expansile lesions. Single myelomas and single metastatic deposits have to be considered particularly in the differential diagnosis. A successful aspiration biopsy often can be obtained. Giant cell tumors may be treated by radiation, if one can be certain of their character, with some success. On the other hand, they are readily and certainly cured by excision and this would seem to be the method of choice. The excision should include a sufficient portion of healthy rib on either side along with the attached muscle, periosteum and the underlying pleura unless the latter be quite free from the tumor.

The ribs are often the site of lesions of multiple myeloma. Two single myelomas of ribs have come under our care. The *x*-ray appearance was similar to the well known picture of the multiple form. If thorough *x*-ray studies of the remainder of the skeleton fail to show another lesion, removal of the apparently single lesion is justifiable in the faint hope that no other focus exists. Negative *x*-rays and indeed negative autopsy findings in such patients do not exclude with certainty other minute foci. In the presence of single or multiple myelomas, the Bence-Jones test for proteinuria may and may not be positive. A negative test is therefore of no value but a positive result is very useful. One of our patients died of pneumonia following operation; the other remained well for eighteen months at which time other lesions appeared. She died nine months later of myeloma.

Metastatic Lesions. The ribs are involved by metastases much more frequently than by primary growths. The commonest source of these metastases is cancer of the breast but any carcinoma giving rise to blood-stream metastases may be responsible. Since these secondary lesions may make themselves evident long before any symptoms are produced by the primary lesions, the recognition of their character is important. They may give rise to local pain or soreness or to pain that is referred along the course of one or more intercostal nerves or in the case of involvement of the upper ribs to brachial plexus pain. On the other hand, extensive metastatic involvement of ribs may be present without any directly related symptoms.

If the *x*-rays show multiple areas of involvement, the condition is likely to be confused only with multiple myeloma. I have, however, seen extensive mottling of the ribs in cases of general debility of unknown origin that resembled very closely the appearance sometimes produced by multiple metastases from breast cancer.

The single lesions may be confused with giant cell tumors or single lesions of multiple myeloma. We have seen single metastases of ribs in a few small and apparently easily operable cancers of the bronchus.

When ordinary methods of clinical examination have failed to disclose a primary cancer from which metastases might have arisen and the diagnosis therefore remains in doubt, aspiration biopsy will often be conclusive. The destruction of the cortex of the ribs has

been sufficient usually to make this procedure a simple one. It may not be possible from the small bit of tissue removed to be certain of the nature of a primary lesion, but it will be possible usually to recognize that it is a secondary cancer and thus save the patient from an unnecessary operation.

When these metastatic lesions are producing pain, the use of deep *x*-ray therapy is worthy of trial. In those arising from cancer of the breast relief of pain is the rule. Those arising from cancer of the bronchus are less certain to respond. Metastases from malignant adenoma of the thyroid apparently vary in their response, some failing to respond at all. On the other hand, I have seen an enormous metastatic tumor of rib from a primary cancer in an adenoma of the thyroid disappear completely and the patient has now been well for several years.

Ewing's Tumors. Ewing's tumor of ribs, if one may judge from the number of cases reported, is a rare disease. Only eight cases in which the diagnosis was certain have been reported. One further case was reported by me in which the diagnosis was not confirmed histologically. The patient developed metastases since that report and died but an autopsy unfortunately was not obtained. The ultimate outlook in all Ewing's tumors, regardless of the location, is bad. The best hope would seem to be early radical removal. Palliation only may be expected from radiotherapy.

Chondrosarcoma. Chondrosarcomas may be divided into two groups: those that are primarily malignant and arise in the absence of a pre-existing benign growth and those that represent late malignant changes in a benign chondroma. If one may judge from the few cases in this series, the primary malignancy is more common. One has the impression that the pathologist's opinion as to the benign or malignant character of these tumors, if based on the histological picture alone, is unreliable. There are, of course, those tumors in which the picture presented is so definitely one of malignancy that no possible doubt need be entertained but there are many others in which the clinical course, *x*-ray appearance and histological characters must all be taken into account if a reasonably accurate opinion is to be formed. From the clinical aspect, the strongest suggestion of malignancy comes from a rapid increase in size. The simple chondromas have usually shown no definite increase in size from the time that they were discovered accidentally and may have been known to exist for a very long period without appreciable change. In the small series that has come under my observation there have been three quite different *x*-ray appearances: (1) a circumscribed shadow barely visible on the *x*-ray films, (2) a circumscribed dense shadow of more or less flocculent character and (3) an extensive destruction of rib such as might be found in any destructive malignant growth. The first two differ in no way, as far as I can determine, from the picture of benign cartilage growths; the third leaves no doubt as to the malignant nature of the lesion.

Early radical excision of chondrosarcomas of a circumscribed character should offer a reasonable prognosis. They must be removed along with adjacent muscles and usually, if not always, the underlying pleura. In the rapidly growing ones such as Case XIV, no form of treatment offers any hope. Radiation is not effective and its use before surgical intervention merely renders the surgical procedure more difficult and more hazardous because of the likelihood of the wound breaking down if it has been made through intensively radiated tissues.

Osteogenic Sarcoma. Osteogenic sarcoma of ribs has been reported very rarely and only one occurred in this series. The reported cases suggest that the condition shows a predilection for the posterior portions of the ribs. The patient's attention may be directed to the growth

by the accidental discovery of a swelling or by pain. The *x*-ray appearance is that of a destructive lesion with varying amounts of new bone formation. The picture presented by those showing considerable amounts of new bone formation differs somewhat, I think, from that of the calcifying chondroma or chondrosarcoma, although they probably cannot be differentiated with certainty either from these or some metastatic lesions of the ribs. Final diagnosis must be made always by biopsy.

In the absence of definite deterioration of general health and evidence of metastases these lesions should be treated by radical surgical removal. Treatment with radiation either as a therapeutic test, in the absence of a definite diagnosis, or in the hope of control when a diagnosis has been established by biopsy, is not in my opinion justifiable if the condition appears to be still localized and capable of surgical removal.

CASE REPORTS

Case I. *Giant Cell Tumor.* A man, forty-nine years old, had complained of pain along the right costal margin for ten months. Clinical examination of the chest was negative, but the screen revealed a tumor of the seventh rib in the postaxillary line. There was no deterioration in general health. *X*-ray examination showed a fusiform enlargement and area of destruction of the seventh rib about three inches in length. The hemoglobin was 83 per cent, white blood count 10,800, and Bence-Jones protein test, negative. At operation September 19, 1936, the seventh rib, along with the adjacent intercostal muscles and underlying pleura, were removed from the vertebra to the anterior axillary line. Recovery was uneventful. In the gross the specimen consisted of a soft grayish-red mass, expanding and largely destroying the rib over an area 8.5 cm. long. The cut surface was vascular and dark red brown. The central part appeared to be broken down and was enclosed by a shell of a maximum thickness of 2 mm., the outer part of which consisted of periosteum with a very thin incomplete layer of bone beneath it. Microscopically, the wall of the cystic space was seen to be formed in large part by fibroblastic tissue, which suggested an inflammatory lesion. Other areas presented what seemed to be definite evidence of tumor formation with here and there a few giant cells. Under the thickened periosteum was a thin layer of new bone. A diagnosis of giant cell tumor was made. (Fig. 1.)

The patient died in February, 1939, and though no autopsy could be obtained it was presumed that the cause of death was bronchogenic carcinoma.

Case II. *Myeloma.* A woman, sixty-three years old, had consulted her physician frequently during the previous six to seven years because of pain in the region of the right shoulder. It was considered, apparently without any real evidence, to be due to a mild pleurisy. In the past year she had had a persistent, nonproductive cough and, for six months, had noted an increasing pallor. *X*-ray examination of the chest, made six weeks before admission, showed an expansile tumor of the second rib just lateral to the cartilage but with no change in the underlying lung or pleura. Clinically, she was well nourished, somewhat pale, but presented no abnormality apart from a marked sclerosis of the peripheral vessels and a slight thickening below the left clavicle. This swelling appeared to pulsate. *X*-ray films of most of the remainder of the skeleton showed no other areas of destruction. No other primary tumor from which a metastatis might have arisen could be found. The hemoglobin was 75 per cent, white blood count, 5,200 and the differential smear was not abnormal. The Bence-Jones protein test was negative. At operation on June 17, 1930, the soft tissues of the anterior chest wall were turned upward and mesially from an axillary incision. The tumor and the adjoining rib and cartilage, the attached intercostal muscles, and the underlying pleura were removed en masse. The growth extended from the middle of the second costal cartilage to midaxilla, following the line of the second rib. (Fig. 2.) Recovery was uneventful.

The gross specimen consisted of a fusiform mass 8 by 4.5 by 2 cm. It was soft, dark red, and appeared to be a mass of hemorrhagic material enclosed by a fibrous capsule. Microscopic examination disclosed a very cellular tumor, the cells of which varied greatly in size and shape, some being small and round, others

long and spheroidal. Many of the smaller cells possessed dark-staining eccentric nuclei and were somewhat similar to plasma cells. The larger cells presented considerable variation in the amount and distribution of nuclear content. It was regarded as a single myeloma.

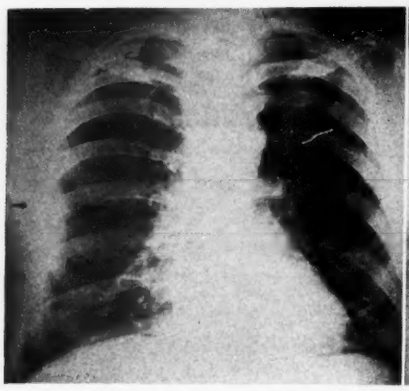

Fig. 1. Case I. A giant cell tumor of the seventh rib.

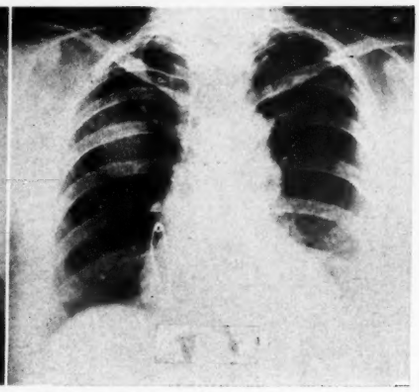

Fig. 2. Case II. A single myeloma of the second rib.

The patient remained quite well until December, 1931, (eighteen months) when an aching pain developed in the left jaw and, shortly afterward, swelling. In January, 1932, she noticed pain above the right knee. *X*-ray films made in February, 1932, showed the typical expansile lesion of myeloma in both these bones. A spontaneous fracture of the femur occurred. She died nine months later. Unfortunately the Bence-Jones test was not repeated.

Case III. *Myeloma.* A man, sixty-one years old, had had a fracture of the eighth rib on the right side three years previously. *X*-ray pictures made at that time showed a fracture of the eighth rib and a normal sixth rib. There had been pain in that region from time to time since the injury, and he had been treated by adhesive strapping. For six months the pain had been more severe, and one week before admission he had noticed a lump. Examination revealed a mass about 5 by 9 cm. along the sixth rib in the right axilla. It was elastic to touch, and there was an impulse on coughing. *X*-ray examination showed no other bony abnormality. The Bence-Jones test was not done. *X*-ray of the chest showed an area of destruction of the sixth rib, beginning about 2 cm. lateral to the costochondral junction and extending along the rib for about 9 cm. The appearance presented was thought to be that of an expansile tumor which had ruptured through the cortex and now lay partly in the soft tissue. At operation, January 15, 1932, the adjacent portions of the fifth and seventh ribs were first resected and the tumor then removed along with the whole of the intercostal bundle on either side and the underlying pleura. The diaphragm was used to close the defect. He died of pneumonia on the third postoperative day. No autopsy was obtained.

Case IV. *Chondroma.* A man, thirty years old, was found to have a tumor of the right chest wall in the course of routine examinations of the chests of a group of factory employees. There were no associated symptoms. The right upper chest was definitely flat, and the retraction involved especially the third rib and cartilage. There was a hard, slightly irregular, fusiform swelling of this rib just beneath the axillary border of the pectoralis major. *X*-ray examination revealed a tumor of the third rib about three inches in diameter which extended laterally from the costochondral junction. (Fig. 3.) It had an irregular mottled appearance. At operation November 2, 1938, an incision was made along the pectoral border and extended medially toward the sternum. The pectoralis major was divided below and reflected upward and inward. The tumor with about one inch of rib lateral to it, a similar length of cartilage medial to it, a small portion of attached muscles, and the underlying pleura were removed. There was only one small band-like adhesion to the underlying visceral pleura, despite the fact that the mass had formed a definite depression in the lung. The second and fourth ribs and cartilages were separated more than usual because of the tumor, and it seemed that a considerable defect in the chest wall would result unless some sort of plastic repair could be accomplished. The fourth rib

and cartilage were split with a motor saw from near the sternum to beyond the point of division of the third rib laterally, leaving the remaining portions of intercostal muscles and periosteum attached to it. The upper half was divided medially, carried up and fastened with a single chromic catgut suture into a seat prepared by removing, subperichondrially, the small remaining portion of the cartilage of the third rib. In doing this, a greenstick fracture was produced at the outer end. The fascia and a thin sheet of muscle were dissected from the pectoralis major muscle and carried beneath the graft to provide a smooth surface between it and the underlying lung. The remaining portion of the pectoralis major was sutured securely into position with chromic catgut, and finally, the subcutaneous tissues and skin were closed. Convalescence was somewhat stormy because of the occurrence of a massive pleural effusion and pulmonary collapse. The patient is now well and has a chest wall of normal stability.

In the gross the tumor was a hard mass of bone and cartilage 11.8 by 7 by 5 cm. Microscopically, it was seen to be made up of large islands of cartilage cells lying in clear spaces surrounded by blue-staining matrix. Many areas had undergone calcification, but true bone formation was not seen. Cyst-like spaces lined by cartilage cells were distributed irregularly throughout the tumor. The surface was covered by a layer of dense fibrous connective tissue. It was regarded as chondroma with calcareous degeneration.

Case v. *Osteochondroma.* A man, forty years of age, had complained of a continuous dull ache or pain in the left chest since the spring of 1916. A lump was noticed in the summer of 1917 over the lower left chest. It had enlarged slowly. There was a rounded swelling extending posteriorly from the costochondral junction of the ninth and tenth ribs and projecting about one inch from the level of the chest wall. *X*-ray examination showed a bony tumor, markedly irregular in outline and appearing to encroach upon the abdominal cavity through the diaphragm. At operation on August 16, 1918, the growth was found to be arising from the ninth rib in the region of the costochondral junction. It was removed along with the neighboring portions of rib and cartilage and the adjacent soft tissue and pleura. The diaphragm was found to have been displaced but not perforated. Recovery was uneventful. In the gross, the specimen consisted of a mass of cartilage and bone the size of an orange. Histologically, it was a benign osteochondroma. The patient is still alive and well.

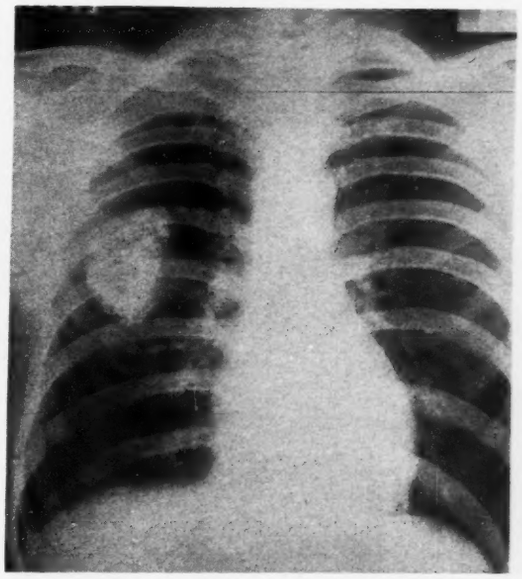

Fig. 3. Case iv. A chondroma of the third rib.

Case vi. *Osteochondroma.* At the age of twelve to thirteen years the patient had a definite injury to her right chest wall. Some time later a lump was noticed in the region of the injury and three to four years after this the lump was removed. It proved to be an osteochondroma. The lump recurred and in 1921, at the age of twenty-four years, she presented herself with an elastic, nodular tumor the size of half an orange, fixed to the right anterior chest wall near the sternum. There were no associated symptoms. On November 27, 1929 the recurrent tumor was removed, this time along with the adjacent portions of the chest wall and the margin of the sternum. It was again an osteochondroma composed almost entirely of cartilage. There has been no recurrence.

This case illustrates the tendency of these tumors to recur following incomplete removal.

Case vii. *Osteochondroma.* A man, fifty-one years of age, first noticed a swelling in the region of the inner end of the left first rib about six months previously. He had taken a plaster mould of it and had satisfied himself that it had enlarged during the past few months. Movement of the arm produced some soreness

in the region of the swelling and a pain which was referred to the shoulder area. Examination revealed a hard mass apparently bony, involving the inner end of the left first rib and cartilage, extending to the sternum and either involving or incorporating the inner end of the clavicle so that it was impossible to be certain whether or not the clavicle could be moved apart from it. *X*-ray showed what appeared to be a bony tumor apparently originating in the region of the costochondral junction of the first rib. There was no apparent involvement of clavicle or sternum.

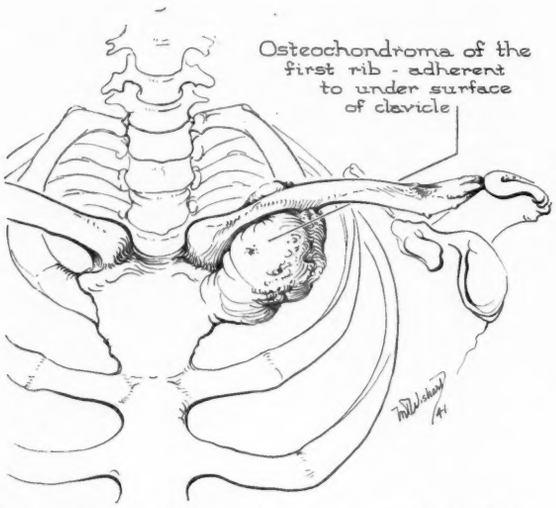

Fig. 4. Case vii. Illustrating the anatomical relation of the tumor.

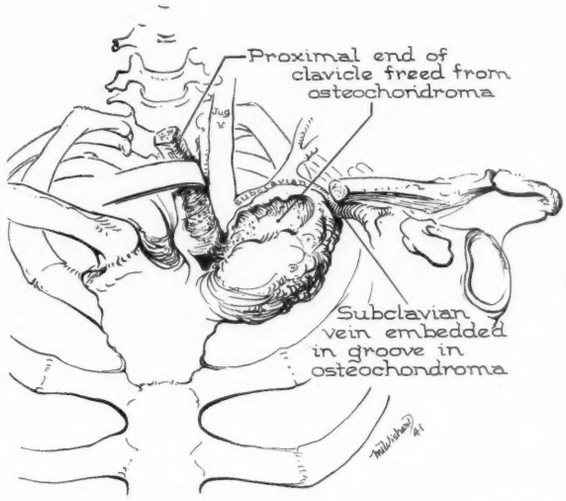

Fig. 5. Case vii. Showing the manner in which the exposure was obtained and the mass freed from the surrounding structures.

Operation demonstrated the fact that removal of a tumor from this particular region involved some special hazards and dangers. A wide U of skin and subcutaneous tissue was turned from the chest wall well upon the base of the neck. The pectoralis major and platysma muscles were divided from the clavicle, leaving sufficient muscle attached that they might be sutured back into position. The clavicle was now divided with a Gigli saw at about its middle so that the inner end might be turned up out of the way. (Figs. 4 and 5.) It was found that the clavicle was either so intimately adherent to the growth or actually involved by it that it seemed unsafe in case the tumor should prove to be malignant, to separate the two. The lower third of the clavicle was therefore chiselled off with an osteotome and left attached to the tumor. The remaining two-thirds being now free was easily turned upward and inward out of the way. The great veins were dissected out, the phrenic nerve retracted and the attachment of the scalenus anticus divided. The subclavian vein was embedded in a U-shaped groove in the tumor and intimately adherent, its lumen being reduced by one-half at this point. It was dissected free with great difficulty. The pleura stripped readily from the inner aspect and remained intact. (Fig. 6.) No line of differentiation could be made out between the mass and sternum and a V-shaped piece of sternum was therefore removed with the mass, the first rib being divided three-fourths inch distal to the tumor. The divided clavicle was re-united by a Kirschner wire in its lumen and a stainless steel wire suture to maintain apposition passed through drill holes made before the division. The muscles were re-united to the clavicle and the wound closed. (Fig. 7.) Recovery was uneventful. The tumor proved to be a benign osteochondroma composed of dense bone with irregular extensive cartilage formation throughout.

Case viii. *Chondroma.* A woman, sixty-four years old, was seen December 8, 1936. Since girlhood she had had a hard swelling of small size on the left chest above the breast. Three years ago this lump had begun to increase in size and recently had been enlarging quite rapidly. It was not painful or tender. Examination revealed a nodular tumor the size of an orange, attached to the left anterior chest wall in the neighborhood of the costochondral junction of the first and second ribs. *X*-ray

revealed a calcifying tumor arising from the first rib in the region of the costochondral junction. It was removed along with the adjacent rib and cartilage on December 21, 1936.

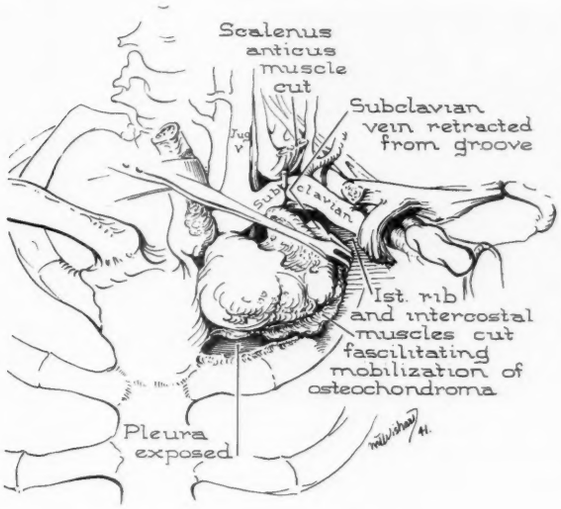
Scalenus anticus muscle cut
Subclavian vein retracted from groove
Subclavian
1st. rib and intercostal muscles cut facilitating mobilization of osteochondroma
Pleura exposed

FIG. 6. Case VII. Same as Figure 5.

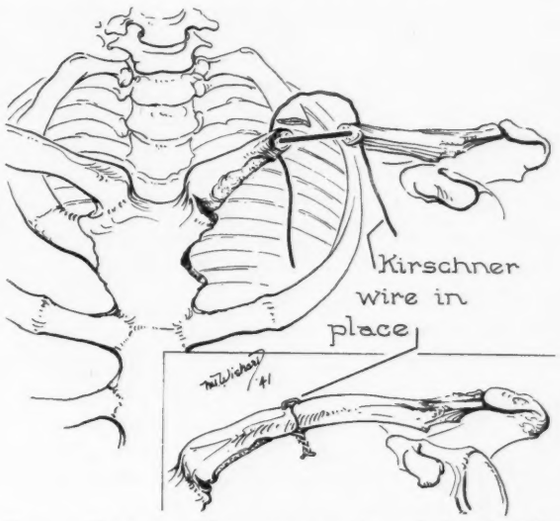

FIG. 7. Case VII. The reconstruction of the clavicle.

The specimen consisted of a nodular tumor 11 by 8.5 by 8 cm. The cut surface was grey in color, smooth and apparently cartilaginous. There was a central area of degeneration and hemorrhage. Microscopic examination showed that the mass was composed of cartilage cells and fibrous tissue with areas of hemorrhage and calcification. It was regarded as a benign chondroma. There has been no recurrence.

CASE IX. *Chondroma.* A woman, sixty-five years of age, was seen July 18, 1939. She had had a swelling over the right anterior chest wall for thirty years. It had slowly increased in size during ten years and during the past five years had grown very fast. Six weeks previously a mild injury had resulted in ulceration. The patient died on July 23, 1939, apparently as a result of infection and debility. The tumor was removed at autopsy. (Fig. 8.) It was 20 by 17 by 14 cm. and on cut section appeared to be cartilaginous with numerous cystic spaces and areas of degeneration and hemorrhage. Microscopically, it was composed for the most part, of mature cartilage in which were areas of fibrous and myxomatous tissue. There was considerable old hemorrhage with dense infiltration of acute and chronic inflammatory cells. The final diagnosis was chondroma with myxomatous degeneration, hemorrhage and infection.

CASE X. *Chondrosarcoma?* A man, aged forty-five years, was admitted in August, 1940. He had noticed a swelling over the right anterior chest wall fifteen years before. It had increased in size gradually over the years but was thought to have grown more rapidly during the past two years. It had produced no symptoms. Examination revealed a somewhat irregular tumor the size of a lemon attached in the region of the costochondral junction of the ninth and tenth ribs. In the *x*-ray the tumor cast a barely visible shadow. Since the patient was really in the hospital for treatment of a very toxic goiter and had been operated upon recently for this, the tumor was removed under local anesthesia on September 16, 1941, no attempt being made by the surgeon to do a wide removal.

The specimen consisted of a hard nodular tumor 8.5 by 7 by 5 cm. The cut surface was greyish in color, smooth and glistening. There was a good deal of calcium. Microscopically, it was made up of atypical cartilage cells that tended to be arranged in groups with a good deal of cartilage matrix between them. There was considerable evidence of necrosis and hemorrhage. It was regarded by the pathologist as a chondrosarcoma but from a clinical point of view was thought to be more probably benign. There has been no recurrence to date.

CASE XI. *Chondrosarcoma.* A man, sixty years of age, was seen on April 3, 1940. A growth was noticed on the right chest wall in August, 1938. There was no pain. The growth increased in size gradually. A biopsy

done elsewhere in October, 1939, was reported as malignant. Examination revealed a tumor the size of a grapefruit involving the ninth, tenth and eleventh right ribs in the costochondral region *X*-ray showed that it was densely calcified. The mass along with the adjacent portions of the chest and abdominal wall was removed on April 9, 1940. The pleura and peritoneum were stripped from the tumor quite readily. Closure of the defect was imperfect. The specimen measured 13 by 12 by 8 cm. with attached portion of three ribs and soft tissues. On cut section it showed hemorrhage, necrosis and irregular areas of calcification. The middle of the three ribs was destroyed entirely over a length of three inches. Microscopic examination showed that the tumor possessed a dense, fibrous capsule freely supplied with blood vessels. Running inward from the capsule were fibrous septa between which were atypical cartilage cells. The central portion showed marked myxomatous changes. Diagnosis was chondrosarcoma. At present the patient is well apart from the presence of a hernia for which a belt must be worn. There is no evidence of local or general recurrence.

Fig. 8. Case ix. An enormous tumor of some thirty years' standing which although apparently benign finally resulted in death from infection that followed ulceration.

Case xii. *Chondrosarcoma.* A man, aged thirty-nine years, was admitted October 27, 1940, with a history of pain and swelling over the left upper chest posteriorly for two years. He attributed these changes to a fall suffered two years before but since the swelling was much the same size at that time as on admission it was believed that the blow merely attracted attention to the condition. One year later he had consulted a radiologist who made a diagnosis of osteogenic sarcoma and gave an intense course of deep *x*-ray therapy. Apart from the production of very severe *x*-ray changes in the skin and deeper tissues of the chest wall this treatment had resulted apparently in no change. His general health was good. Examination revealed a swelling about four by six inches attached to the ribs of the upper chest wall medial to the scapula and overlapping the scapula and vertebral spines but not attached to them. There were intense radiation changes in the skin although no radiation had been given for six months. *X*-ray showed an irregular calcifying mass arising from the fourth rib in the region of the angle and lateral to it and largely destroying the contour of the rib over an area of about three inches. The neighboring ribs seemed merely to be involved in the extension of the tumor. On November 1, 1940, the tumor mass along with the posterior portion of the upper seven ribs, attached soft tissue and underlying pleura were removed en masse. The fourth rib was disarticulated, the others removed to the transverse process only. By using the scapula a fairly satisfactory closure was obtained. The radiated tissues subsequently broke down, the patient developed an empyema and had a stormy postoperative course but is now well.

The specimen consisted of a hard tumor mass 14 by 11 by 6 cm. with attached ribs. Histologically, it was made up of cartilage cells showing great variation in size and staining. The stroma varied from a hyaline to a fibrous type. There were irregular areas of myxomatous degeneration and calcification

throughout the tumor. Diagnosis was chondrosarcoma with calcification.

Case XIII. *Chondrosarcoma.* A man, sixty-three years old, noticed a swelling of the left chest wall in October, 1937. The mass was not painful and was noticed by chance. *X*-ray films made shortly after showed a tumor about 10 by 12 cm., arising from the region of the costochondral junction of the third rib on the left side. The shadow was very indistinct because of an almost complete absence of calcium. Examination revealed a flat, firm swelling about two inches in diameter arising from the third left rib lateral to the costochondral junction. What appeared to be the whole growth was removed by his surgeon on November 16, 1937.

The specimen consisted of a mass 7 by 6 by 5 cm., which appeared to surround the rib. It was moderately firm, pinkish and somewhat lobulated. On section it was gelatinous in appearance and very friable. Microscopically, the tumor was seen to be made up of large masses of cartilage cells. Near the periphery it was very cellular, and the cells were less differentiated in character and were invading between the fibers of attached muscle. There were a few areas of myxomatous degeneration.

The patient received one series of 3,000 R. postoperative radiation but failed to report for further treatment. He returned November 3, 1938, with numerous secondary masses in the region of the incision. Subsequent radiation failed to control these recurrences and he died of the disease.

Case XIV. Chondrosarcoma. A sixteen year old Jewish boy was admitted to the hospital on February 4, 1941, with a three-day history of dry cough, left-sided pleural pain and shortness of breath. Three months previously his chest had been *x*-rayed, because his tuberculin test was found positive in a routine survey and the report was negative. Examination revealed a well nourished boy with a thoracic scoliosis with convexity to the right. There was a peculiar prominence of the ninth and tenth ribs on the left side posteriorly which he said had been present all his life. There was a left pleural effusion which on aspiration proved at first to be clear fluid and later to be blood tinged. The fluid was sterile and no tumor cells could be found in it. There was a low-grade fever and a leucocytosis of 16,200. *X*-rays showed a destructive lesion with some calcium deposition in the ninth rib on the left side beginning medial to the angle and extending for a distance of three inches laterally. A biopsy was taken. This showed a lesion made up of atypical cartilage cells growing in a haphazard fashion with numerous mitotic figures. There was no bone formation.

The patient's course was down hill rapidly and he died on March 13, 1941.

Case XV. *Osteogenic Sarcoma.* A man, fifty-four years of age, was admitted July 21, 1938. One week previously his son had noticed a deformity of the lower chest. There were no local symptoms, and the general health had remained good. There was an elongated swelling of the eleventh right rib which extended forward for about seven inches from a point near the spine and was of a maximum width of about two and one-half inches. It was firm, somewhat irregular in outline and not tender. *X*-ray examination showed replacement of the eleventh rib by a circumscribed tumor which had a vacuolated appearance suggestive of either a chondroma or chondrosarcoma. At operation, July 22, 1938, an ample incision was made in the long axis of the tumor. The tenth rib was first resected subperiosteally to give access, and the involved rib was then removed from anterior to the tumor to and including the head of the rib, along with the attached intercostal muscles and underlying pleura. The muscles were closed over the defect. Postoperative recovery was uneventful and at present there is no clinical or *x*-ray evidence of recurrence.

In the gross the specimen was bony hard. The attached muscle tissue was intimately adherent to it. Microscopically, the mass was made up of dense sheets of cells with long spindle-shaped nuclei and varying amounts of cytoplasm, growing in irregular fashion. Distributed throughout these cells were many multinucleated bone cells about which there were deposits of new bone. It was regarded as an osteogenic sarcoma.

SUMMARY

Tumors of the chest wall comprise all growths affecting tissues of a similar character elsewhere in the body. While they do not present any special pathological characters they do, because of their location, present special surgical problems. The differentiation between benign and malignant growths in the fibrous and cartilage

groups is, as elsewhere, difficult and cannot be done on histologic appearances only. When operated upon by a surgeon unaccustomed to treating thoracic lesions, these tumors are perhaps likely to be removed incompletely because the surgeon fears to open the chest widely. They should be operated upon always with a closed type of anesthesia, preferably of the intralaryngeal or intratracheal type. The anesthetist as well as the surgeon should be accustomed to handling thoracic cases. Radiotherapy cannot be expected to have much effect on most of the primary conditions and should be used as a rule, only when surgical removal is impossible.

Because they occur rather infrequently and are the most interesting of the group, case reports of the primary lesions of ribs and cartilages have been given in some detail.*

* Cases I, II, III, IV, V, XIII and XV were reported previously in *The Journal of Thoracic Surgery*, 9: 145–159, December, 1939.

A DEMONSTRABLE enlargement of the thyroid gland is present in almost all patients suffering from acute thyrotoxic disease, but not in all. In rare instances the gland is not obviously enlarged, but even in these a definite increase in size can be demonstrated at operation. One should not hesitate to diagnose exophthalmic goiter without a palpable thyroid if the other signs warrant.

RELATIVE FREQUENCY AND SITE OF PREDILECTION OF INTRATHORACIC TUMORS*

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PRIMARY intrathoracic tumors are common. They may originate in the lung, in the mediastinum or from the various elements of the chest wall. By far the most important and frequent intrathoracic neoplasms are tumors of the lung, which are more properly termed bronchiogenic tumors because it has been established that practically all lung tumors are of bronchial origin and rarely arise from the lung parenchyma. Second in frequency are the various tumors of the mediastinum, and, third, the relatively rare neoplasms which begin in the chest wall and invade the thoracic cavity.

BRONCHIOGENIC TUMORS

Bronchiogenic tumors have until recently been considered a rare form of disease. During the past ten years, however, it has become more and more apparent that the incidence of endobronchial neoplasms is increasing and that bronchiogenic carcinoma is one of the most common forms of cancer. At the present time, carcinoma of the lung is of about the same frequency as carcinoma of the colon, and constitutes between 5 and 10 per cent of all cancers. Koletsky[1] in a study of 7,685 consecutive cases studied at autopsy found bronchiogenic carcinoma second only to carcinoma of the stomach. Kikuth,[2] Hirsche and Halpert,[3] Matz,[4] and many others, at postmortem observations, have found carcinoma of the lung either second or third in frequency, being preceded only by carcinoma of the stomach and carcinoma of the large bowel.

Another important observation has been made in various series of autopsy studies, namely, that more recent autopsy reports demonstrate a progressive increase in the frequency of pulmonary tumors. For example, Weller[5] at the University of Michigan found carcinoma of the lung occurred in 0.1 per cent of the first 1,000 autopsies performed at that institution. In the second 1,000 autopsies, the incidence was 0.5 per cent, and in 450 cases of the third 1,000 cases studied, the incidence was 0.8 per cent. Holzer[6] reports that in autopsy studies conducted between 1895 and 1904, only 1.04 per cent of all carcinomas discovered at postmortem were bronchiogenic in origin. Corresponding data from 1915 to 1924 showed an increase to 6.9 per cent. Ochsner and DeBakey[7] have further emphasized the steady increase of bronchiogenic carcinoma by a review of the death rates per 100,000 population in the United States who have died of malignant tumors of various kinds and are recorded by the mortality statistics of the Bureau of Census. These data show that from 1920 to 1936 there was no appreciable increase in the incidence of carcinoma of the stomach, uterus or gall bladder, whereas there was a marked increase in the incidence of carcinoma of the lung. The same authors have recently reported that in their experience carcinoma of the lung has increased approximately five times in the past seven years. This statement was based on a study of necropsy material at the Charity Hospital in New Orleans. Brines and Kenning[8] in a study of 936 patients with various types of tumors found carcinoma of the lung fourth in frequency. At the Barnes Hospital Chest Service, sixty-two proven cases of bronchiogenic carcinoma were seen in a

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three-year period from 1933 to 1936. During the following three years, 117 bronchiogenic carcinoma cases were admitted for treatment. It becomes apparent, therefore, that the amazing increase in the incidence of bronchiogenic carcinoma cannot be attributed to improvements in diagnosis or increased awareness of the condition, but must be considered actual as demonstrated both by autopsy material and clinical observations.

It is now generally agreed that practically all pulmonary tumors arise from the bronchi. There has, however, been much confusion in classification of lung tumors because of the uncertainty of exact cellular origin and multiplicity of names which have been applied to the various lesions based on their microscopic appearances. This controversy does not, however, include true squamous cell cancer of the bronchus. Like squamous carcinoma in other locations, this variety of cancer occurs more frequently between the ages of forty and sixty, and is predominantly a disease of males. Other bronchiogenic carcinomas have been designated as round cell carcinomas, oat cell carcinomas, adenocarcinomas, depending upon the various arrangements of the cells. It is beyond the province of this paper to discuss in detail various controversial conceptions regarding classification of pulmonary tumors. However, it is necessary to mention briefly a few of the recent conceptions of pulmonary tumor formation to avoid confusion in the subsequent discussion of the localization of these tumors.

Endobronchial tumors which are sometimes classified as benign are rare as compared to true bronchiogenic carcinomas. The most common of this group is the so-called bronchial adenoma. These tumors may arise in any portion of the bronchial tree, but are more commonly found in the larger bronchi. The anatomic origin of bronchial adenomas may, therefore, be considered identical to that of bronchiogenic carcinomas. Unlike squamous cell carcinoma of the bronchus, these tumors occur frequently in females and may appear at any age. Brunn and Goldman[9] report an incidence of 64 per cent in females in forty-two collected cases.

The tumors may be in the shape of a polyp attached to the bronchial mucosa by a small pedicle, but more often the intramural type is present where only a part of the tumor presents endobronchially. Not uncommonly, these tumors assume a dumbbell pattern, with the greatest portion of the neoplasm extending outside of the bronchus into the parenchyma of the lung. Brunn and Goldman[9] and others believe that the bronchial adenoma has such a distinct difference in appearance and growth potential that it deserves to be classified in an entirely different category from true bronchiogenic carcinoma. There is, however, a sharp difference of opinion on this point, and others, notably Womack and Graham,[10] believe that many endobronchial tumors, including bronchial adenomas, have a common origin, and are the result of the failure of embryonic bronchial buds to develop in a normal way. When the entodermic elements in the embryonic bud predominate, the tumor may be considered a bronchial adenoma, alveolar carcinoma, adenocarcinoma, etc. Mesodermic domination will result in what has been called by others chondroma, sarcoma, etc. Their conception is that these tumors should be considered potentially malignant, although they may be benign when first recognized. Frequently, these tumors resemble the mixed tumors of the salivary glands and it is not unusual to find in them cartilage and even bone. When malignant they manifest a markedly invasive quality but often without microscopic evidence of great activity of growth. Halpert[11] explains the formation of endobronchial tumors on the basis of what he terms reserve cells. These are undifferentiated entodermal cells capable of developing into ciliated epithelium, cylindrical cells, goblet cells, or may differentiate into any kind of epithelium that an entodermal cell is capable of producing. Another common classification of bronchio-

genic tumors is: (1) Squamous cell carcinoma; (2) small cell or undifferentiated cell carcinoma; (3) endocarcinoma. The confusion regarding classification of bronchiogenic tumors by the microscopic appearance does not, however, affect certain important facts concerning their site of predilection. For clinical purposes, all bronchiogenic tumors can be divided into two groups: (1) Tumors of peripheral origin, in which the lesion originates in minor bronchi, or from the periphery of the lung; (2) neoplasms originating in large or major bronchi.

Tuttle and Womack[12] have pointed out the differences in symptomatology and prognosis, depending upon whether the tumor originates in the periphery of the lung or near the pulmonary hilum. They have shown that tumors originating in the major bronchi are more slowly growing, probably because of the tough cartilage which must be penetrated if the neoplasm spreads by direct extension. Tumors of large bronchi produce symptoms earlier because obstruction or partial obstruction of the bronchus will result in atelectasis or pulmonary suppuration much earlier in the course of the disease than if the tumor is situated in a small peripheral bronchus.

In slightly more than half of the cases studied by Tuttle and Womack[12] the tumor was situated in a major bronchus. Boyd[13] and Edwards[14] found a somewhat higher incidence of centrally located tumors, the former finding only 10 per cent of the tumors located in the periphery, and the latter, 20 per cent.

In 216 patients with bronchiogenic carcinoma reported by Gebauer,[15] squamous cell carcinomas were situated in the major bronchi in 70 per cent of the cases. Tumors classified as adenocarcinoma arose in secondary bronchi in 90 per cent of the cases, and in the main bronchi, in only 10 per cent. Sixty-six per cent of tumors classified by Gebauer[15] as small cell carcinomas began in the main stem bronchi. Controversial opinions concerning histologic classifications make evaluation of these data difficult. It is apparent, however, that in general the more slowly growing and better differentiated carcinomas have a tendency to arise in the major bronchi, whereas the rapidly growing, undifferentiated varieties of cancer usually originate in the smaller bronchi.

The relative frequency of involvement of the right and left lung does not vary enough to be of great practical importance. Fischer[16] studied 3,735 cases and found the right lung involved in 53 per cent of the cases, the left in 45 per cent, and a bilateral lesion overriding the carina in 2 per cent. Ochsner and DeBakey[7] collected 4,732 cases from the literature, and found right-sided involvement in 58.4 per cent, and left sided tumors in 41.6 per cent. In 784 of Fischer's[16] cases, 18 per cent of the tumors originated in the right main bronchus, and 14 per cent of the tumors were in the left main bronchus. The most frequent site of origin was in the right upper lobe bronchus, where approximately 19 per cent of the tumors were situated. The left upper lobe bronchus was the site of origin in 16.4 per cent of cases, the right lower lobe in 16 per cent, the left lower lobe in 13 per cent, and only in approximately 0.2 of the cases was the middle lobe bronchus involved. It is interesting to note that the superior divisions or dorsal divisions of the lower lobes are an infrequent site of tumor formation. In a series of 117 cases at the Barnes Hospital, we have seen only one instance of bronchiogenic carcinoma originating in this bronchial segment. This finding is particularly interesting because the superior divisions of the lower lobe are probably more frequently involved in lung abscess formation than any other portion of the lung.

Posterior Superior Sulcus Tumors. Tumors originating in the posterior superior sulcus of the thorax at the apex of the chest often produce a rather typical symptom complex consisting of unilateral pain in the shoulder girdle, Horner's syndrome, and paresis of the hand. Roentgenograms will usually show a mass confined to the posterior superior sulcus of the chest when these

symptoms are present. Pancoast[17] first described the symptom complex associated with the abnormal roentgenographic shadow, and it became the custom to refer to the condition as a Pancoast tumor. Ray[18] and others who have studied the so-called Pancoast tumor and its associated symptom complex are convinced that in the great majority of cases, the symptoms and roentgenographic findings are the result of invasion of the pleura by an ordinary bronchiogenic carcinoma and that neurofibromas, or any other tumor situated in the posteromesial portion of the chest, are capable of producing the same symptoms. It would seem, therefore, more proper to regard the term, Pancoast tumor, as a topical classification rather than a pathologic entity and to mention the condition in connection with bronchiogenic carcinomas.

RARE CONDITIONS WHICH SIMULATE PULMONARY TUMORS

Tuberculomas or calcified pulmonary abscesses may be indistinguishable from true pulmonary neoplasms until they are removed. Graham and Singer[19] reported three such cases in 1936, and later Haight[20] reported a calcified pseudotumor of the lung which proved to be a tuberculoma. Thoracic aneurysms may also be confused with intrathoracic neoplasms, although usually the true nature of the lesion can be determined by roentgenological examination. Graham[21] has reported two instances of nonpulsating aneurysms of the ductus arteriosus in which the positive diagnosis could be established only by exploration of the thorax. In two other cases at the Barnes Hospital, thoracic exploration was necessary to establish a positive diagnosis of aortic aneurysm in which pulsation could not be determined by fluoroscopy or by roentgen kymograms.

MEDIASTINAL TUMORS

Tumors of the mediastinum may be benign or malignant, but, except for rather rare cystic types of tumors, practically all mediastinal tumors are potentially malignant. The site of origin of mediastinal neoplasms is rather constant for each group, and a convenient method for describing them is to divide the various types into: (1) Tumors which usually originate in the anterior mediastinum; (2) posterior mediastinal tumors; (3) a miscellaneous group of comparatively rare neoplasms which may be located in any part of the mediastinum.

ANTERIOR MEDIASTINAL TUMORS

Lymphomas. A common variety of anterior mediastinal tumor arises from lymphatic tissue, and may be classified under the inclusive term of lymphomas. This group includes lymphosarcoma, Hodgkin's disease, so-called leukosarcoma, thymomas or any neoplasm which may originate in the lymphatic tissue situated in the mediastinum. Differentiation of the tumors of the lymphoma group from other types of anterior mediastinal tumors before treatment is undertaken is of primary importance because, in general, tumors composed of lymphatic tissue will react to roentgenotherapy and are not amenable to surgical extirpation. It is, therefore, the custom of many experienced thoracic surgeons to submit all patients with anterior mediastinal tumors to a test of roentgenotherapy. If the neoplasm decreases in size, the futility of surgical intervention is established. Confusion and controversy regarding the origin and relative frequency of tumors which arise from the lymphoid tissue in the mediastinum make a reliable estimate of the relative frequency of the various types of lymphomas impossible. Baldridge and Awe[22] have a workable classification for lymphogenous tissue, recognizing as the Hodgkin granuloma a sclerosing type of lesion, the lymphoblastic type as lymphosarcoma and the endothelial type as lymphoepithelioma. The lymphocytic type, they consider a manifestation of leukemia with or without blood changes. Ewing's[23] classification is based on three anatomic elements, namely, lymphocytes,

reticulum cells of the follicles and pulp, and endothelial cells. It is, however, often impossible to classify histologically the lymphomas of the anterior mediastinum which do not have other general manifestations of disease of the lymphatic system, or in which tissue is not available for biopsy. The same difficulties exist when one attempts to classify tumors of the thymus gland.

Fig. 1. Lateral roentgenogram showing anterior position of a teratoid tumor of the mediastinum.

Heuer and Andrus[24] have been able to collect 230 cases of malignant thymomas. In their series of 145 cases of mediastinal tumors, studied at the New York Hospital, 32 per cent were considered lymphomas. Of these, eighteen were diagnosed lymphosarcoma, and twenty-nine as Hodgkin's granuloma. In a series of eighty-seven cases of mediastinal tumors studied at the Barnes Hospital, in thirty-eight instances the tumor was classified as a lymphoma. In this group there was no other evidence of disease of the lymphatic system which could be demonstrated by involvement of other lymph-nodes or by changes in hemograms. In all instances the lesion diminished in size when roentgenotherapy was given.

Dermoids and Teratomas. Harrington[25] employs the term, teratoid tumors, for all dermoids and teratomas of the mediastinum. He feels that it is more accurate

than the inclusive term, dermoid tumors, because the majority of these neoplasms are derived from all three germinal layers.

FIG. 2. Photograph of teratoma of the mediastinum after successful surgical removal.

The structures of the teratoid tumors vary from thin walled cysts filled with cloudy fluid, or which may contain ectodermal derivatives such as skin, hair, or teeth, to solid tumors, depending upon the varieties of tissue which predominate. Heuer[24] has stated that the teratoid mediastinal tumors are the most common neoplasms of the mediastinum, but at the Barnes Hospital Chest Service, we have found that tumors of the lymphoma group occur more frequently than the teratoid variety. With the exception of the lymphomas, however, the incidence of teratoid tumors was more than twice that of any other kind of mediastinal neoplasm. Of 233 collected cases, including twenty-five at the Barnes Hospital, in only three instances has the tumor been reported to be in a posterior position in the mediastinum. These were in the cases of Krause,[26] Von Torok,[27] and of Smith.[28] In a series of 116 cases studied by Hedblom,[29] all of the tumors were in an anterior position. The mass was predominantly right-sided in sixty-three instances, and more apparent on the left in fifty-three cases. The teratoid tumors are thought to take their origin from cell rests probably of branchiogenic elements which are drawn into the thorax by the descent of the heart and diaphragm. (Figs. 1 and 2.)

Mediastinal Thyroids. Thyroid tissue situated in a substernal position may produce an anterior mediastinal tumor which cannot be diagnosed definitely until the mass has been removed. In the great majority of cases, however, involvement of the thyroid tissue in the neck is rather obvious and will indicate the true nature of the lesion. Total or complete thoracic goiter may occur without cervical goiter, but this condition is rare.

Cysts of the Mediastinum. Thin walled cysts containing fluid have been given a variety of names including cystic hygromas, cystic lymphangiomas, and ciliated epithelial cysts. Cysts of the respiratory epithelium are often called bronchiogenic cysts. Lambert[30] has recently introduced convincing evidence that many cysts of the mediastinum arise from the pericardium through the defects in the process of its development. Forty-one instances of mediastinal cysts classified as either cystic hygromas, bronchiogenic cysts or cystic lymphangiomas are recorded in the literature. Five cases seen at the Barnes Hospital in which operation was performed successfully have been reclassified and are considered to originate from the pericardium, since Lambert's[30] conception of their origin has been known.

POSTERIOR MEDIASTINAL TUMORS

The most important group of neoplasms which arise from the posterior mediastinum are the primary nerve tumors. These neoplasms take origin from any of the elements of nerve tissue and may, therefore, be more precisely classified as ganglioneuromas, neurogenic sarcomas, neurofibromas, sympathicoblastoma, etc., depending upon the cellular element which predominates. Harrington[25] has employed the term perineural fibroblastoma in describing fourteen primary nerve tumors which he has treated surgically. He justifies the term perineural fibroblastoma by the fact that in each

instance the tumor was thought to arise from the sheath of the nerves. Thirteen of the group were benign tumors and one was considered malignant. Practically all primary nerve tumors have the common factor of being located in the posterior mediastinum and of being malignant or potentially malignant. Not infrequently, tumors which arise from the spinal or sympathetic nerves involve not only the posterior mediastinum but the vertebral canal, and assume an hour-glass or dumbbell shape.

Fig. 3. Lateral roentgenogram showing usual posterior position of a primary nerve tumor.

Difficulties in exact histological classification make it impossible to evaluate exactly the incidence of each type of primary nerve tumor which is recorded in the literature. Including thirteen cases treated surgically at the Barnes Hospital, 135 instances of neurogenic tumors of the posterior mediastinum are recorded. Twenty-two of these are considered neurofibromas, seventy ganglioneuromas, nineteen are considered neurinomas, (recorded by Heuer and Andrus)[24] and the remaining twenty-four as perineural fibroblastomas or simply as primary nerve tumors. Not infrequently, neurofibromas of the mediastinum may be associated with other stigmata of Von Recklinghausen's disease. This was the

case in one of our thirteen cases, and the condition has been recorded frequently in the literature. (Figs. 3 and 4.)

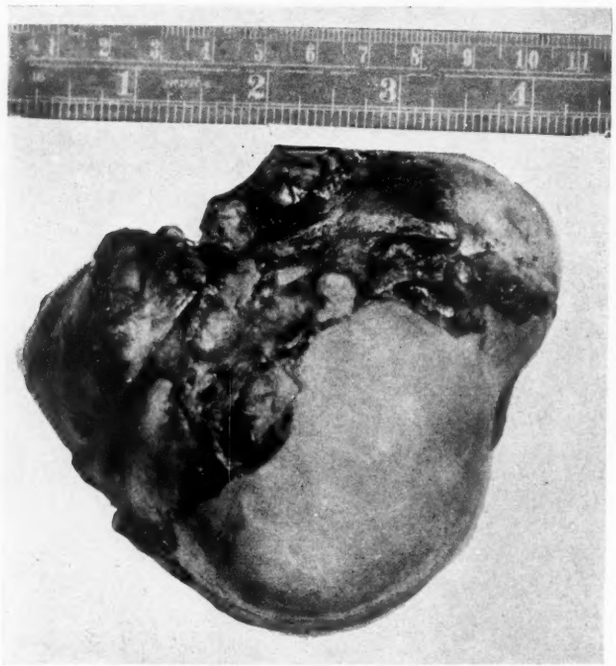

Fig. 4. Photograph of neurofibroma of posterior mediastinum after successful removal.

RARE MEDIASTINAL TUMORS

Mediastinal Fibromas and Sarcomas. Thirty-two cases of fibroma of the mediastinum have been reported. In many instances, however, the diagnosis could be questioned, and it seems likely that confusion with fibrosarcoma or neurofibromas had occurred in many instances. The same difficulty arises when one attempts to evaluate the miscellaneous group of tumors included under the diagnosis of sarcoma of the mediastinum. These may arise from the malignant degeneration of chondromas, primary nerve tumors, or from any of the connective tissue elements of the lymph-nodes or of the thymus. In our experience, it is often impossible to make an exact histologic classification of rapidly growing mediastinal sarcomas. In many instances, however, the positions and clinical aspects of the neoplasms have suggested that their origins were either from nerve tissue or the periosteum of the ribs.

Carcinoma of the Mediastinum. Since it has been appreciated that pulmonary carcinoma is common and often invades the mediastinum, fewer cases of carcinoma of the mediastinum have been reported. There is evidence that the thymus gland is the most common source of primary carcinomas of the mediastinum. In many instances, however, it is impossible to establish the exact origin. By far the most common cause of invasion of the mediastinum with carcinoma is extension or metastasis from a primary bronchiogenic tumor.

Mediastinal Tumors Arising from Bone and Cartilage. Chondromas, osteochondromas, chondromyxomas, enchondromas or chondromyxosarcomas may take their origin from the costal cartilages, sternum or spine and present in the mediastinum. All varieties are rare. Chondromas are more common in the anterior mediastinum arising from the costal cartilages, and all others of the group are more commonly located in a posterior position. Heuer[24] was able to collect only fourteen cases of mediastinal tumor which might be considered to originate from bone or cartilage.

Intrathoracic Cysts of Intestinal Structure. Nicholls,[31] of London, has reported the successful removal of an intrathoracic stomach and was able to collect twelve cases of intrathoracic cysts of intestinal structure from the literature. Graham[32] at the Barnes Hospital has had two cases of intrathoracic stomachs, one in an infant, and the other in a young man in whom the tumor of the mediastinum was discovered during an examination for admission to service in the army. In this case, the intrathoracic stomach was removed successfully, and the patient has now been admitted to military service. Two explanations have been advanced for the development of intrathoracic tumors of intestinal structure: (1) That they are cells from the foregut which remain in the thorax during the course of development; (2) that the cells are derived from the vitello-intestinal duct. In all of the cases reported by Nicholls[31] and in Graham's[32] two cases, the tumor was located in the posterior mediastinum. Twelve of the fifteen known cases were right-sided.

Lipomas of the Mediastinum. Intrathoracic lipomas may occur in a variety of forms, but are comparatively rare. Their anatomic location may be of three types: (1) Tumors in which an intrathoracic mass is continuous with an extrathoracic extension, causing the neoplasm to assume an hour-glass shape; (2) thoracic lipomas which extend into the neck; (3) lipomas which lie entirely within the thoracic cage. McCorkle et al.[33] have been able to record only thirty-four instances of thoracic lipoma from the world literature, including a case of their own. At the Barnes Hospital we have seen only two cases of lipomas of the mediastinum.

Mediastinal Xanthomas. Another unusual type of intrathoracic new growth has been called xanthoma by some, and sarcoma by others. The characteristic histologic finding in xanthomatous neoplasms is the xanthoma or foam cell. Phillips[34] recorded two cases which he had observed and collected 3 similar cases from the literature. He considered this type of neoplasm to be nonmalignant and advised discontinuance of the term sarcomas in describing them.

TUMORS ORIGINATING IN THE CHEST WALL

It is frequently difficult to determine from published accounts of intrathoracic tumors whether their origin was actually in the mediastinum or from the chest wall. It is understandable why this situation can exist because it is often difficult to determine this point at operation or autopsy when the tumor is of large size. Tumors may arise from the ribs, the pleura or the nerves in the chest wall and manifest themselves as intrathoracic masses.

Benign intrathoracic tumors arising from the chest wall are rare. Harper[35] was able to collect sixty chondromas, nine fibromas, seven osteotomas, and eleven benign giant cell tumors reported in the world literature. These tumors usually do not invade the thorax but present as external masses. The tendency for all chondromas or osteochondromas of the chest wall to undergo malignant degeneration has been well established, and many believe that a large proportion of cases of intrathoracic sarcomas begin as benign chondromas or neurofibromas.

A great variety of tumors which may arise from the pleura have been described. These may be benign or malignant, primary or metastatic. Fibromas, lipomas, myxomas, angiomas, various types of sarcomas, including myxosarcoma, round cell, spindle cell and fibrosarcoma have been described. All of these tumors are rare and require no particular comment.

Epithelioma of the pleura is probably a misnomer. Robertson[36] has shown that primary malignant tumors of pleural tissue are probably always sarcomas and that the great majority of so-called endotheliomas of the pleura represent extension or implantations of carcinoma from another primary source, usually the lung.

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TUMORS OF THE THYMIC REGION AND MYASTHENIA GRAVIS

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THE etiology of myasthenia gravis is unknown and the functions of the thymus gland are unknown, therefore, it is apparent that one enters a field of speculation if one attempts to consider the relationship of abnormalities of the thymus to myasthenia gravis. It is known that myasthenia gravis is a disease characterized by abnormal fatigability of muscles, usually by characteristic lymphocytic infiltration of these and other organs and frequently by failure of involution or neoplasm of the thymus. It is known that benign tumors of the thymus are rare and that most of those that have been reported have been present in patients with myasthenia gravis. The frequent association of the two would appear to be more than a coincidence.

The first description of a thymic tumor in association with myasthenia gravis was that by Weigert[1] in 1901. Bell,[2] in 1917, collected from the literature an account of fifty-six cases of myasthenia gravis in which either an operation or an autopsy had been performed and twenty-seven of these exhibited abnormalities of the thymus. A diagnosis of thymoma was made in ten and the remaining seventeen patients were considered as having either a persistent or an enlarged thymus. Norris[3] restudied the problem in 1936 and found that there were thirty-five lesions of the thymus in a total of eighty necropsy reports on patients with myasthenia. He expressed the opinion that pathologic changes may be found in the thymus in cases of myasthenia gravis in direct ratio to the care with which they are sought. In 1939, Blalock, Mason, Morgan and Riven[4] found eighteen additional cases in the literature and reported one of their own. Thus, up to 1940 there had been reported fifty-four instances of abnormalities of the thymus in approximately 110 autopsies or operations on patients with myasthenia gravis. Additional cases have been reported since that time. Miller[5] described the autopsy findings on five patients with myasthenia gravis, two of whom had an encapsulated tumor of the thymus and two had a persistent thymus. Undoubtedly, a good many cases have not been reported. For example, Dr. E. H. Campbell of Albany, New York, has removed recently typical thymomas from two patients with myasthenia gravis.

The presence of an abnormality of the thymus should be suspected in every patient with myasthenia gravis. Definite enlargements or tumors may be demonstrated by fluoroscopic or *x*-ray examination. A film taken in the lateral position is usually best for demonstrating the lesion.

One cannot make dogmatic statements concerning the effects of the removal of thymomas upon the course of myasthenia gravis. This is due to two reasons: In the first place, there have been too few such operations to allow critical judgment. The most striking result is that reported by Blalock, Mason, Morgan and Riven.[4] This patient, previously a severe myasthenic, has been well now for five years following thymectomy. In the second place, spontaneous remissions are not uncommon and apparent beneficial postoperative effects may be explainable on this basis.

The operation is a relatively simple one. An upper median sternotomy gives an excellent exposure and the tumors can usually be shelled out without difficulty. It is obvious that either the patient should

be in a remission at the time of the operation or one should be able to control the disease by the use of prostigmin or some other drug. Since myasthenia gravis is a disabling disease and since the operative procedure is not a very grave one, it would appear that the encouraging experiences indicate the advisability of the surgical removal of clinically demonstrable thymic tumors in patients with myasthenia gravis.

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Usually in thyrotoxicosis the tendon reflexes are exaggerated. In the beginning and in milder cases they are normal. Late in the disease the reflexes are sometimes lost but in that event other causes for their absence should be sought.

INTRATHORACIC GOITER

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THE fact that certain goiters are partly or wholly intrathoracic is now accepted as a serious and not uncommon pathologic condition. When one considers that many of these goiters are completely hidden in the bony thorax, however, it is not surprising that only in recent medical times has much been known of this disease. Intrathoracic goiter was first described anatomically by A. Haller in 1749.[1] It was much later, in 1830, when C. Lingl[2] gave the first clinical description of this lesion.

M. Dubourg,[3] in 1826, recorded an instance of strangulation and death due to intrathoracic goiter, and in 1828 A. Adelman[4] reported a case of malignant disease in this type of goiter. The term, "goiter plongeant," was coined by Malard in 1851[5] to apply to those substernal adenomas which popped up into view above the sternum with increased intrathoracic pressure. The monograph, "Die Struma Intrathoracica," by F. Wuehrman, in 1896[6] was the most comprehensive study to that date.

The recognition of intrathoracic goiter in the earlier years was, of course, limited by the lack of roentgen diagnosis until 1899. At this time Schieff[7] added another most important milestone to the understanding of this disease by showing the value of the *x*-ray in the diagnosis of intrathoracic goiter.

It is interesting today to note that the classical descriptions of exophthalmic goiter by Parry, Graves, and Basedow, as quoted by Major,[8] failed to include any case in which the symptoms and signs were those of a goiter extending into the thorax below the sternum. This finding is in accord with present day experience that the thyroid gland in typical exophthalmic goiter rarely, if ever, extends deeply into the thorax. One cannot but wonder if these writers saw no cases of hyperthyroidism due to a substernal adenomatous goiter which were difficult of explanation.

Considerable confusion has existed in descriptions of intrathoracic goiter because of different nomenclatures used by various authors. Inclusion of the terms, "substernal" and "retrosternal" and "subclavicular," under the single grouping of "intrathoracic" goiter seems amply justified and has been fairly well accepted. From 12 to 30 per cent of all goiters could be so classified according to various reports.[1,4,8,9] Some authors, however, prefer to limit the term, "intrathoracic," to those goiters which extend down to or below the arch of the aorta. If this more rigid definition is followed, incidence of intrathoracic goiter ranges from less than 1 to 10 per cent of all goiters operated upon.[10,11,12]

Intrathoracic goiter arising from ectopic thyroid tissue deep within the mediastinum is quite rare. The case of Means[11] in which the mass rested on the diaphragm may fall into this category. From embryologic considerations such glands should lie in close relationship to the aortic arch or possibly the tracheal bifurcation. They may or may not be connected with the thyroid proper by a fibrous cord.[1]

Ptosis of the normal thyroid gland, or a slight dipping down of cervical goiter behind sternum or clavicles should not be classified as intrathoracic goiter, since only part of such goiters are at best subclavicular.

Intrathoracic goiters develop when a single adenoma or one or more of the nodules of a multiple adenomatous goiter descend through the superior thoracic strait into the bony thorax. Most frequently intrathoracic goiters arise from adenomatous goiters, but occasionally a single discrete adenoma which once lay in the lower pole of one thyroid lobe will

descend into the thorax. The descent of these single or multiple nodules into the thorax depends first on their location in the gland. Obviously, a discrete nodule located in the superior pole of the thyroid gland would not find its way into the chest. When, however, the nodule is present in the lower portion of either of the thyroid lobes or of the thyroid isthmus, it may, as it grows in size, readily descend through the superior thoracic strait. One of the factors that influences this descent is the rate of growth which usually is so slow that it requires years for the goiter to enter the thorax. It is, of course, occasionally true that a low-lying discrete adenoma in a young person will have a hemorrhage into it, and since the least resistance to its expansion is in the downward direction, it will so descend into the chest. Whether the growth of these adenomas of the thyroid is rapid or slow, there is nothing but loose cellular tissue to restrict their descent into the thorax through the superior strait. Expansion in other directions is limited by muscles, fascia, vessels or bone. The descent of these low-lying adenomas is furthermore aided very markedly by the contractions of the sternomastoid muscles and of the prethyroid muscles that accompany motions of the head and acts of respiration and deglutition and to some degree by the force of gravity itself.

ANATOMY

The anatomical relation of intrathoracic goiters is most important, not only in considering the effects of these thoracic masses upon adjacent structures but also in the surgical management of their removal. It must be recognized that all, or practically all, intrathoracic goiters lie in the closest association to the trachea, and this relationship to the treachea is one of the important factors in their diagnosis and in their surgical management. It is perfectly obvious, when one considers the origin of the intrathoracic masses from an adenomatous enlargement of the thyroid gland surrounding the trachea, that the intrathoracic portion of the goiter may lie either to the right, to the left, in front of, or behind the trachea. Furthermore, in some cases the adenomatous mass may entirely encircle the trachea with extensions running backward from each lobe. Most commonly, intrathoracic goiters extend from the lower pole of either the right lobe or the left lobe of the thyroid directly down along the trachea into the superior mediastinum. Less common, in fact quite unusual, is the type in which adenomatous nodules grow backward between the branches of the inferior thyroid artery and then go downward behind the trachea. This type is occasionally overlooked in goiter operations because the intrathoracic portion lies out of sight behind the thyroid lobe in the neck.

The arterial supply to intrathoracic goiters comes from the same arteries that supply the normal thyroid gland. Both the superior and inferior thyroid artery have considerable slackness due to their winding course from the major arterial trunk to the thyroid gland. This is most important, not only because it permits portions of the thyroid gland to descend with their arterial supply intact but also because it permits control of the blood supply of intrathoracic goiters by controlling the major thyroid arteries in the neck. The superior thyroid artery, after it leaves the external carotid artery, goes up the neck somewhat and then curves downward to join the superior thyroid pole. The inferior thyroid artery, after it leaves the thyroid axis of the subclavian artery, goes up the neck in quite a wide arch, and after passing beneath the carotid artery and the jugular vein at a level just below the superior thyroid pole, descends along the gland and enters it in the lower or middle portion of the lobe. It is apparent from this distribution of these two major arteries that if structural changes in the thyroid gland occur, and the lobe or part of the lobe descends into the chest, there is sufficient laxity in these arteries to permit them to descend with the lobe. Certainly there is nothing in their

anatomical arrangement which at the start of the descent of the goiter into the thorax exerts tension to hinder this process. The veins coming from the thyroid gland empty in large part into the internal jugular vein, and it seems to be possible for these veins also to elongate as part of the thyroid lobe descends into the thorax. Almost never have we had any significant venus bleeding from within the thorax when a discrete adnomatous mass was lifted out of the chest. The veins all come up with the mass and lead into the jugular vein at a level which usually is readily seen in the operative wound in the neck. These anatomical arrangements of the arteries and veins are most important, then, in the surgical removal of intrathoracic goiters.

The effect that the descent of thyroid adenomas into the thorax has upon the recurrent laryngeal nerve will vary, of course, with the location of the thyroid mass before it starts downward into the chest. It is, nevertheless, true that in most instances, in spite of the presence of deep intrathoracic goiters, there is no vocal cord paralysis. In the past it has been said that when vocal cord paralysis was present one could be quite certain that malignant degeneration of the intrathoracic mass had occurred. This, however, we now know to be a mistake, because we have several times seen preoperative paralysis of one cord in the presence of large substernal goiters in which there was no evidence of malignancy. Whether the substernal goiter pushes the recurrent laryngeal nerve backward as it descends, or in occasional cases in which the goiter descends between branches of the inferior thyroid artery, presses it forward, will of course depend on the point of origin of the goiter. It is our experience, however, that in most cases the recurrent nerve is pushed backward and is well out of the way of the surgeon at the time of the operation.

PATHOLOGY

Most intrathoracic goiters, as we have said, arise from a multiple adenomatous goiter, or the so-called nodular goiter. Less often they result from the descent into the thorax of a single discrete adenoma. Whichever of these two basic pathological factors is present, the intrathoracic goiter is subject to the same degenerative changes that may occur in a goiter which is entirely cervical in location.

Hyperplasia of the intrathoracic thyroid tissue can occur, and in fact, not infrequently does. This results in the development of toxic symptoms, and we have a clinical picture here very little different from that seen in toxic adenomatous goiter when it is in the usual position in the neck. Degenerative changes may occur in the substernal mass with necrosis, hemorrhage and cyst formation. Occasionally, such cysts become very large and produce very serious pressure symptoms. Calcification in varying degree may occur in the intrathoracic goiter, presumably because intrathoracic goiters are goiters which have been present for many years. In some cases malignant degeneration occurs just as it does in nodular goiter in the neck. The malignancy may be diffuse, invading all the structures and going outside of the thyroid capsule, or it may be localized within a discrete adenoma. It may be of any of the types that one sees in thyroid pathology in the neck.

PHYSIOLOGY

Changes in the physiology due to the presence of an intrathoracic goiter are related first to the possibility that hyperthyroidism has developed because of hyperplasia in the intrathoracic mass. This hyperthyroidism is in no way different, in our experience, from the hyperthyroidism that comes from nodular goiter in people of middle life and beyond. Its presence, of course, adds distinctly to the risk of removal of the intrathoracic goiter, just as the presence of hyperthyroidism adds appreciably to the removal of any large goiter.

The changes in the physiology which are specifically related to the anatomical posi-

tion of the goiter in the thorax are all changes which are due to the presence of an intrathoracic mass, and depend on the pressure of this mass on the surrounding structures. The trachea is the earliest organ to show the effect of this pressure. It is deviated from its midline position by the presence of the substernal mass. If the substernal mass lies on both the right and the left side of the trachea, then it is, of course, narrowed by this bilateral pressure. If the goiter lies behind the trachea, the trachea may be broadened laterally and at the same time its lumen narrowed anteroposteriorly by the enlarging goiter forcing it against the sternum. A similar condition may arise when the goiter lies in front of the trachea and pushes the trachea backward toward the spine. The degree of tracheal pressure will, of course, depend on the size of the substernal goiter and the amount of tracheal deviation present. In some cases the trachea becomes so narrowed that the patient has a constant stridor on inhalation. In certain instances the patient finds that there are certain positions of the head in which pressure on the trachea is very much increased and he consciously avoids these positions. Thus we have patients who cannot sleep with their head bent sharply forward because this obstructs their breathing. Others cannot lie on one side or the other in bed because again the trachea is bent over the substernal mass and is definitely narrowed by this mechanical change in its position. It is interesting to observe that very rarely does a substernal goiter affect the course of the esophagus.

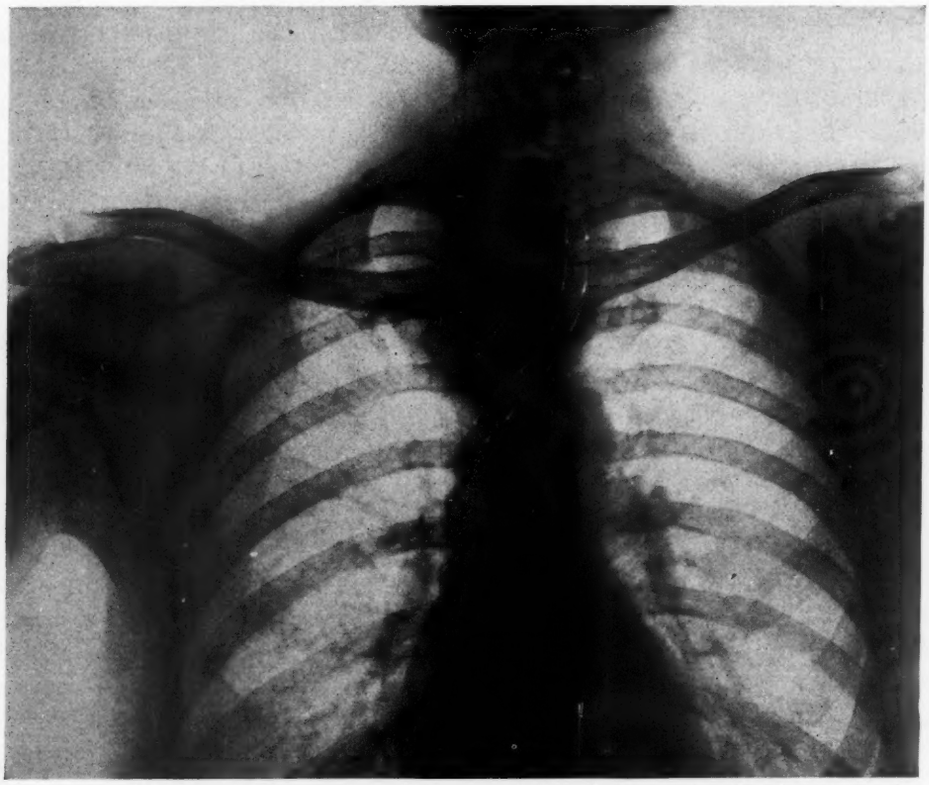


Fig. 1. (L. Y.) This anteroposterior *x*-ray exposed for soft tissue visualization shows the trachea bowed to the left and narrowed to less than half normal size by the pressure of a single large intrathoracic adenoma arising from the right lobe of the thyroid. Tilting of the larynx away from the affected side is well demonstrated.

Physiological effects from the presence of an intrathoracic mass rarely make any changes on the recurrent laryngeal nerve or other nerves in the neck. In a few instances, however, unilateral vocal cord paralysis has occurred in our experience probably due to stretching of the recurrent

laryngeal nerve. The presence of this paralysis in the recurrent nerve may not be recognized by the patient and will be found only by routine examination of the vocal cords. In other instances, however, goiter patients consult the physician because of the fact that they have recently developed hoarseness and this may be the first symptom of a substernal goiter.

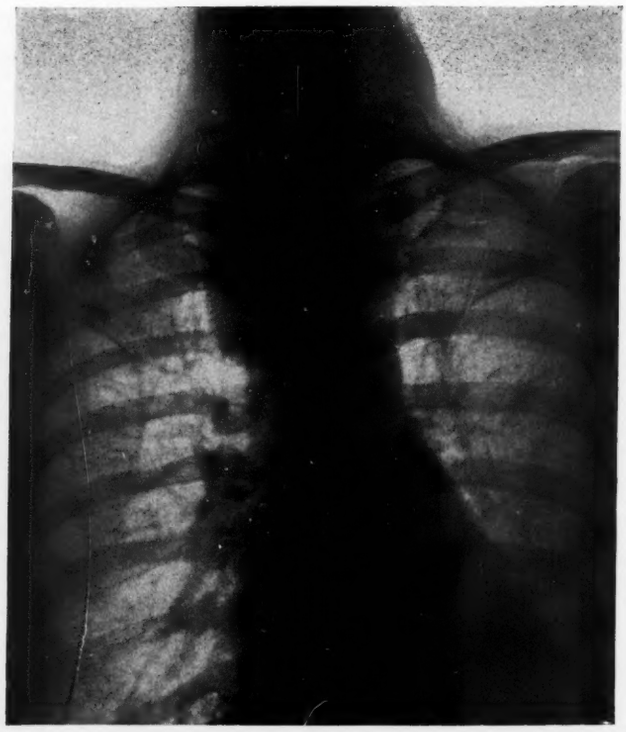
Fig. 2. (M. R.) An obliquely lateral *x*-ray in this case demonstrates deviation of the trachea backward with compression by an intrathoracic goiter at level of the manubrium sterni. At one point the trachea is narrowed to one-third normal diameter. The size of the soft tissue mass is clearly shown.

In patients who have very large intrathoracic goiters, there is sufficient pressure of the goiter against the venous structures coming down through the superior thoracic strait to produce definite engorgement of the veins in the neck and over the chest wall. Such venous engorgement is very marked in certain large goiters, and yet, curiously enough, in other deep intrathoracic thyroid tumors there will be little or no venous engorgement. The venous engorgement may be so marked in some individuals that puffiness of the face and duskiness of color are distinctly noticeable and very large dilated veins are plainly visible over the neck and upper anterior chest wall.

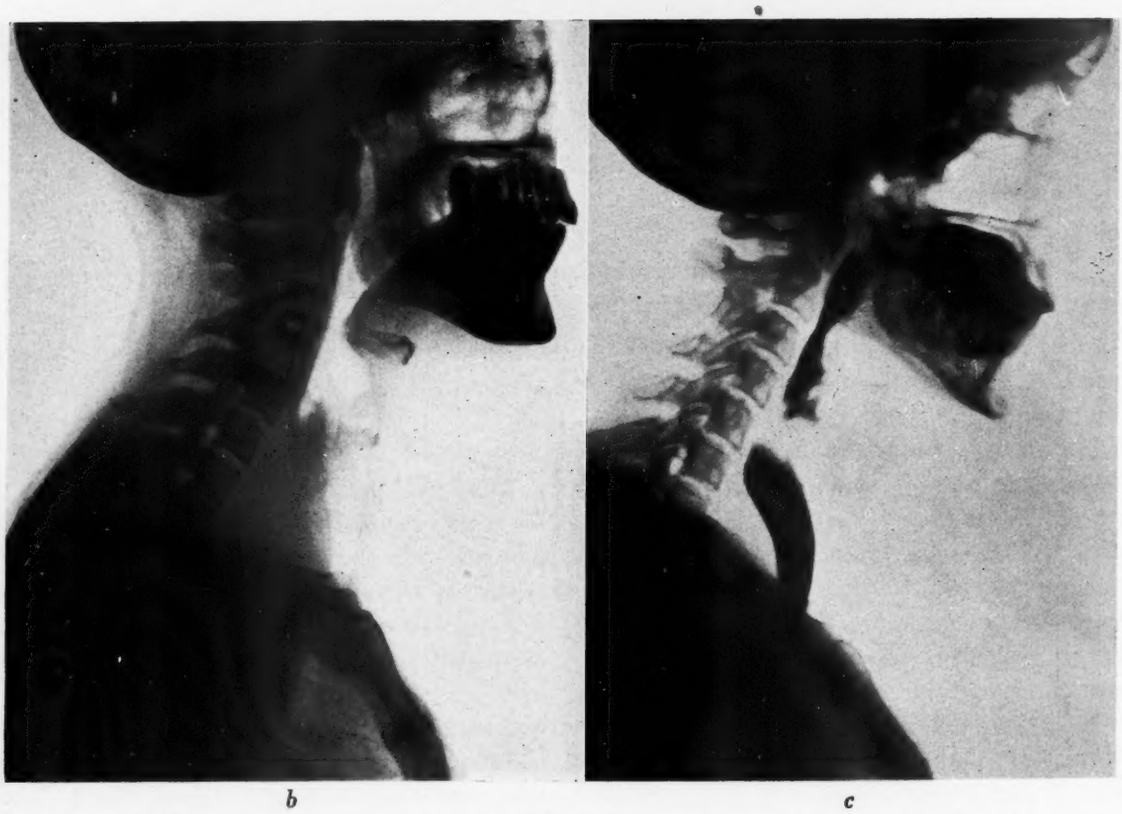
DIAGNOSIS

The diagnosis of intrathoracic goiter can be made, not infrequently, from careful consideration of the patient's history, but in many instances it is made only by clinical and *x*-ray examination. The clinical symptoms that are suggestive of intrathoracic goiter are varied, but most of them are the result of pressure of the intrathoracic mass on the trachea, on veins or on the recurrent laryngeal nerve.

Occasionally, a patient is seen who says that she had a goiter many years ago, but that it "disappeared." When a definite sizeable mass in the area of the thyroid



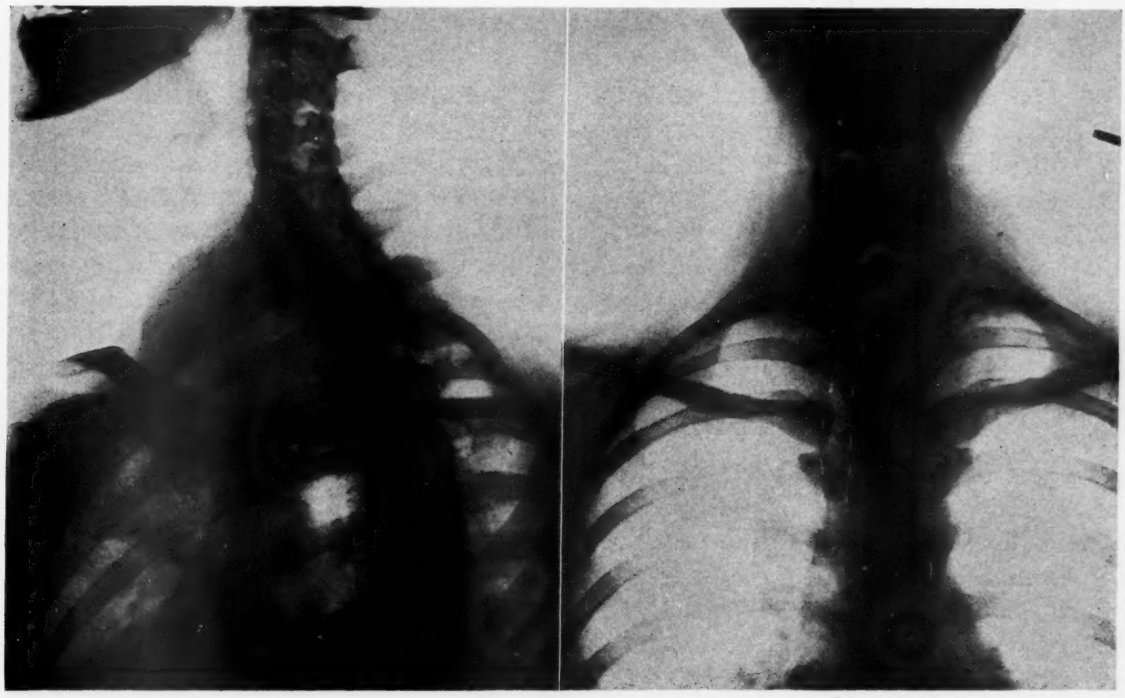
a



b *c*

FIG. 3. (M. C.) In the anteroposterior *x*-ray (*a*) of this patient, the trachea is barely visualized, but apparently nearly in the midline. A soft tissue mass is shown extending below the aortic arch, particularly on the right. A lateral film (*b*) demonstrates marked anterior deviation of the trachea with narrowing of its lumen. Barium swallow (*c*) indicates that the goiter lies behind the esophagus also, this structure being pushed forward with the trachea.

"disappears," one may be very suspicious that it has descended through the superior thoracic strait and so passed from view.



a *b*

FIG. 4. (A. F.) Deviation of the trachea backward and to the right by the pressure of an intrathoracic goiter are shown (*a*) in the lateral and (*b*) anteroposterior *x*-rays. These are the usual views taken in *x*-ray diagnosis of intrathoracic goiter.

More commonly the patient's history states that there has been an increasing sense of pressure in the throat. Occasionally, this pressure takes the form of a lump in the throat which is noticeable when solid foods are swallowed. In well developed intrathoracic goiters the patient notices very definitely the stridor and difficulty of breathing that accompany narrowing of the trachea.

When the intrathoracic mass lies on one side of the trachea and pushes the trachea well away from the midline, the patient will not infrequently state that when she lies with her head on the side at which the goiter is, she has obstruction in breathing; and when she lies with her head on the side away from the goiter, there is less trouble in breathing. The mechanics of this arrangement are perfectly logical and readily understandable.

In some patients the development of hoarseness, and not infrequently the discovery of paralysis of one vocal cord by a physician, leads to the search for a possible substernal goiter. In other instances the gradual development over a period of years of increasingly large veins at the base of the neck and over the upper chest, likewise show the possible presence of substernal goiter.

In some individuals the development of hyperthyroidism, in the absence of any apparent goiter in the neck, may lead the physician to search for a substernal goiter. The presence of real hyperthyroidism with no palpable enlargement of the thyroid gland in the neck should make any physician consider the possibility of an adenomatous goiter beneath the sternum in the mediastinum.

The physical signs that accompany the presence of substernal goiter are varied. In the normal individual one can, with considerable practice, palpate the upper pole and the lower pole of both the right and left lobes of the thyroid. If the thyroid is

enlarged, this palpation of the poles is much simpler. If then, in the presence of an adenomatous goiter, one can feel the superior pole on each lobe, and the inferior pole on one side, but is quite unable to get beneath the inferior pole and feel its lower borders on the other side, he at once becomes suspicious, if not certain, that there is a substernal extension and an intrathoracic goiter.

In some patients there is a discrete adenoma beneath the sternum which rises in the neck when the patient coughs or sneezes or in any other way increases the intrathoracic pressure. This ability of a discrete adenoma in the superior mediastinum to rise up into the neck is not commonly seen but when present is diagnostic. Intrathoracic goiters, when one can palpate their superior borders or watch them with the fluoroscope, move up and down with deglutition just as do goiters in the neck. This movement with deglutition helps to differentiate the intrathoracic goiter from intrathoracic dermoids or other tumors.

We have never found percussion of the superior mediastinum and increase in the dullness of this area of great help to us in the diagnosis of substernal goiter. Most substernal goiters show, in some degree at least, in the neck, or can be palpated in the sternal notch or just to one side beneath the sternomastoid. When such a mass is felt and when it rises with deglutition, percussion may help the examiner in deciding that there is a larger mass beneath the sternum.

The diagnosis of substernal goiter can almost invariably be made by satisfactory and adequate *x*-rays of the superior mediastinum. The *x*-ray technician must understand that his pictures must be sufficiently soft to show the outline of the trachea, and the patient must have the trachea filled with air and hold his breath while the picture is taken. It is very important that not only anteroposterior views of the trachea and the superior mediastinum are taken, but also that lateral views as well are obtained.

A discrete adenoma lying just beneath the manubrium of the sternum and directly on the trachea may make no great impression on the anteroposterior picture of the trachea, and certainly the trachea will not be deviated either to the right or the left of the midline. However, a lateral view of the trachea in this same case will show its marked deviation posteriorally, due to the presence of this goiter in front of it. A similar situation is obtained when the goiter is behind the trachea and pushes the trachea forward rather than laterally. In some instances *x*-rays are taken while the patient is swallowing a barium mixture, in order to show the relation of the esophagus to the trachea and to the goiter. This has been very helpful to show the position of the intrathoracic mass. In many cases of intrathoracic goiter, the goiter itself will not show in the *x*-ray and its presence will be known only because of its deviation from the normal position in the midline of the trachea. In other cases, however, when the goiter is very large or when it contains much calcified material, the actual shadow of the goiter itself will be visible; and as *x*-ray technicians become more expert in their tracheal pictures, the actual shadow of the goiter is more likely to appear in the plates.

TREATMENT

The treatment of intrathoracic goiter is surgical removal, and it is unnecessary to state that the earlier in the course of development of a substernal goiter surgery is undertaken, the easier, simpler and safer is the surgical procedure.

It has been surprising to us to note in many instances the size of intrathoracic goiters that can be removed through the superior thoracic straits without cutting the sternum or freeing the sternoclavicular articulation. In fact, the senior writer in his entire experience has never had occasion to enlarge the bony thoracic strait for the removal of a substernal goiter, nor has he yet encountered such a large noncancerous intrathoracic goiter that it could not be re-

moved without enlarging the bony thoracic straits.

The anesthesia for the removal of intrathoracic goiters is, of course, of the utmost importance. Here particularly is it necessary to have an anesthetist trained in goiter surgery, and adept in handling the specific problems that arise when a trachea is distorted in position and narrowed in its caliber. We now prefer, and routinely use, intratracheal anesthesia with cyclopropane and oxygen for anesthetic agents. This is the most satisfactory arrangement in our experience. The only objection to it, and it is not a serious one, is that the intratracheal catheter is somewhat stiff and somewhat resistant when one is attempting to dislocate the intrathoracic mass; and it does not bend and give as much as the trachea would if the catheter were not inside of it. However, the fact that anoxemia cannot occur and that the patient's respiratory process is under the control of the anesthetist, far outbalances the slight mechanical objection to the catheter in the trachea.

The first requirement in the surgical removal of an intrathoracic goiter is a wide incision. The usual transverse cervical incision is made above the clavicle, the skin flap lifted and the muscles split in the midline longitudinally. The sternomastoid is then freed from the prethyroid muscles on each side, and the prethyroid muscles are clamped and cut on each side, even though the substernal goiter is present on only one side. This cutting of the muscles on the uninvolved side is not necessary in small tumors, but in large growths it is very helpful, because it gives a great deal of freedom for the lobe of the thyroid on that side to move when the manipulations for the extirpation of the goiter in the chest are going on.

When the field is thoroughly exposed by wide cutting of the prethyroid muscles and thoroughly dry, a careful exploratory finger is placed along the posterior surface of the capsule covering the intrathoracic goiter. Gentleness is most important at this stage, and as the finger sweeps around the capsule and comes forward, one meets the venous trunks which run from the goiter to the jugular vein. If the finger is gently hooked onto these, they can be caught with snaps, cut and tied at once. This relieves a great deal of the bleeding which might otherwise be present if the goiter were quickly extirpated. In some instances it is desirable to catch, cut and tie the superior thyroid artery in order to gain greater freedom of motion of the involved lobe of the gland. This, however, is by no means always necessary.

With the veins now tied, the finger is gently swept around the substernal mass keeping most carefully and gently in the proper line of cleavage. If one gets away from this, hemorrhage occurs or one breaks into the pleural cavity. Therefore, it is most important that adequate time and gentleness be employed at this stage to free the capsule of the mass from the adherent surrounding tissue. In some cases, at this stage of the procedure, the goiter can be somewhat lifted forward and the inferior thyroid artery seen, caught, tied and cut. In other cases, however, one cannot get at the inferior artery until the mass is fully delivered from the chest.

When the intrathoracic mass is delivered into the neck, a wet gauze pack is immediately slipped down into the cavity from which it came. This is done to prevent any sudden expulsive cough from causing undue pressure on the pleura and rupture of the pleural cavity, and also to control any ooze from nearby veins that may be occurring. With the goiter now delivered into the neck and in clear vision, the course of the inferior thyroid artery can usually be seen, and occasionally the recurrent laryngeal nerve can also be seen and more thoroughly avoided. Usually, however, the nerve is not seen, and one proceeds at once with the removal of the intrathoracic mass. Due care is taken to avoid injury to the trachea which one must recall may well be distorted by the delivery of the tumor. Also, great care must be taken to inspect the surface of the mass for parathyroids and to protect

these and leave them behind as one proceeds with the excision of the goiter.

When the intrathoracic goiter is removed and hemostasis is complete, a cigarette drain with a very small amount of gauze protruding is placed into the intrathoracic cavity from which the goiter came. Almost never has it been necessary for us to pack this cavity with gauze, but a drain is desirable because not infrequently serosanguineous fluid will collect in the cavity and have to be released if drainage is not instituted at operation. It is our custom to start pulling the drain out slowly on the fourth day and to have it completely removed on the sixth or seventh day, unless undue amount of discharge is present.

As we have said before, most substernal goiters can be delivered intact without any rupture of the capsule that surrounds them. This is made possible by gentle, slow dissection with the fingers which finally encompass the entire goiter, and then the slow deliverance of the goiter by a finger beneath the lowermost pole. Occasionally, however, the goiter is so large that it cannot be brought through the superior thoracic strait intact, and one can then readily put a finger through the capsule at the upper portion of the intrathoracic mass and rupture it, permitting the soft, jelly-like contents to ooze out. When this has happened, the mass becomes much smaller and then almost invariably can be teased up through the superior thoracic strait. The bleeding which accompanies this rupture of the capsule is not great nor troublesome and is readily controlled when the entire tumor is delivered and the major blood vessels are caught and tied.

CONCLUSIONS

Intrathoracic goiter is not an infrequent disease and one with possibly serious effects if not recognized and treated sufficiently early.

Certain features of the history and physical examination usually suggest the presence of intrathoracic goiter. The *x*-ray offers important and frequently diagnostic information by revealing deviation and compression of the trachea and less often the soft tissue mass causing this.

With rare exceptions intrathoracic goiters may be removed safely by appropriate surgical methods.

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CARCINOMA OF THE LUNG AS A SURGICAL PROBLEM*

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A SOLID front must be developed among forces fighting a disease which gives quarter for a short time in its life history and then invariably causes death. All members of the profession must be on the alert at all times for cancer which may still be in its early stage. Cancer of the lung is no exception. We cannot be held to task so definitely for the lives of patients who have metastatic disease when they first consult us. For that group our educational program may be at fault. There are also those unfortunate few whose malignant lesion progresses to the incurable stage without the production of symptoms. The profession cannot escape its responsibility, however, to that large group of patients who come to us for help while their pulmonary cancer is still confined to its original site.

From a practical point of view right now, doctors can help patients with cancer of the lung more effectively by first, speeding up the mechanism of discovery; second, agreeing on proper management for the patient who has either a suspected or proved cancer; third, appraising the condition of the patient properly as to operability; and fourth, recognizing the value of surgical exploration in patients presenting presumptive evidence of cancer but in whom the diagnosis is unverified histologically. It is the purpose of this paper to discuss primary cancer of the lung as a surgical problem with particular reference to these four points. A résumé will be given of the results of a clinical study in 174 patients suspected of having pulmonary carcinoma in whom tissue verification was obtained in 127.

PROGRESS ANTICIPATED IN EARLY DISCOVERY

When contributing factors to early discovery of cancer of the lung are weighed in the balance, the scales tip well toward the hopeful side. There are four factors upon which an optimistic point of view is based. (1) The frequency of cancer of the lung will force the profession to be constantly on the lookout for it. (2) Early telltale symptoms do develop which will give the first doctor consulted a clue as to a possible organic chest lesion. (3) The chest lends itself well to contrasting shadows on the *x*-ray film and will yield presumptive evidence of an early lesion. (4) In the earliest stages the majority of the growths can be diagnosed with certainty. Three-fourths of the lesions originate in one of the main bronchi and are visible and can be biopsied bronchoscopically.

General interest in cancer of the lung is being forced upon the profession by the established fact of its frequency. Malignancy in this location is now near the top of the list as a cause of death from cancer. Even as late as five years ago, autopsy statistics seemed unbelievable when it was reported that carcinoma of the lung accounted for 10 per cent of all deaths caused by cancer, and was exceeded only by lesions which originated in the stomach and colon. An absolute as well as relative increase in the frequency of cancer of the lung is generally conceded. Ochsner and DeBakey,[1] after making a survey on the relative frequency of various types of cancer, stated, "During the period 1920–1936, inclusive, the incidence of cancer of the stomach and duodenum, of the liver and gall bladder, and of the uterus and of the skin showed little, if any increase. The incidence of cancer of the lung showed a progressive rise throughout the United States." They found the death rate for carcinoma of the lung in 1936 to be 3.6 per hundred thousand, whereas in 1920 the rate was only 1.1. These authors also reported that cancer of

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the lung as found at necropsy in the Charity Hospital in New Orleans had increased five times in the past seven years. In 1938, as a cause of death, cancer of the lung even exceeded cancer of the stomach in frequency. Together with the numerical increase in cases coming under clinical observation, the literature on the subject has swelled tremendously. No doctor will be permitted to forget that the lung is a common site for malignancy.

The first factor then which will speed up the mechanism of early discovery will be the development of a full appreciation of the possible presence of malignancy in any patient complaining of chest symptoms. In the past there has been too long an interval between the time the doctor first saw the patient and the time when he began to suspect that there might be a serious organic lesion. In our series of cases, records show that the average interval between the time of the first visit to a doctor and the first *x*-ray was six and one-quarter months. This interval can be shortened to a matter of days or weeks, instead of over one-half a year.

SYMPTOMS AND PHYSICAL SIGNS NOTABLY UNRELIABLE

What is the clue to possible early cancer of the lung? The great majority of patients who harbor primary malignancy of the lung do have symptoms which point the diagnostic finger at the chest. Ninety-eight per cent of the patients in our series complained of some chest symptoms, and the great majority of the chest symptoms came early in the course of the disease.

The study of frequency and sequence of symptoms by various authors has resulted in common agreement on the following points: (a) There is no characteristic symptom complex in carcinoma of the lung. The condition may simulate many other diseases of the lung. (b) Chronic cough is by far the most frequent and therefore the most important of all symptoms. If there has been an habitual cough for years, a change in the character and frequency of the cough is significant. Eighty-seven per cent of the patients in our series complained of cough. (c) Attention may be first directed to the chest by symptoms of a respiratory infection of a chest cold. Persistence of symptoms, signs, or *x*-ray changes may be indicative of bronchial occlusion and tumor. (d) Important, but less frequent symptoms, are hemoptysis, chest discomfort or pain and wheezing. Gastrointestinal disturbances may be present especially if the left lower lobe bronchus is involved. (e) Dyspnea, weight loss and symptoms related to accumulation of fluid in the pleural cavity are late signs. Therefore, such symptoms need not be discussed in a consideration of early diagnosis.

It has been our experience that carcinoma of the lung has been mistaken for tuberculosis more frequently than any other pulmonary disease. Other erroneous diagnoses were also made, such as unresolved pneumonia, bronchitis, asthma, pleurisy and empyema.

Just as there is no definite symptom complex in early malignancy of the lung, there are no distinguishing physical signs. In fact, small peripherally situated growths are notable for producing no abnormal signs. Stem bronchus tumors occluding but one of the lobular divisions may not produce abnormal physical signs. When a main bronchus or one of its lobar branches is involved, variable signs are produced depending upon the degree of obstruction and the presence or absence of secondary infection. It should, therefore, be emphasized that physical signs in early carcinoma of the lung are variable and unreliable. Their absence should not excuse the first doctor consulted in arranging for a chest *x*-ray. The presence of physical signs, suggesting congestion, consolidation, fluid, unresolved pneumonia, bronchitis, asthma or tuberculosis does not necessarily rule out malignancy. Any abnormal physical sign or any persistent symptoms relating to the chest call for further investigation.

CHEST ROENTGENOGRAMS ESSENTIAL

The structure of the lung creates an unique position for it among internal organs in respect to early detection of cancer. The density of the expanded lung is less than any other soft tissue organ. Therefore, it lends itself better than any internal organ to *x*-ray scrutiny. Minute lesions in peripheral portions of the lung field will cast a shadow. Small stem bronchus lesions whose direct shadow is obscured by trunk markings soon occlude a major bronchus or one of its subdivisions. Either lobar or lobular atelectasis results, which gives itself away on the chest film. In our experience, the *x*-ray revealed some abnormal shadow, either the discrete shadow of the tumor, an enlarged hilar shadow, or altered density due to atelectasis in 96 per cent of the cases. Discrete shadows of the tumor itself or massive atelectatic shadows are so striking that the doctor is usually induced to institute further investigation immediately.

It should be emphasized, however, that as more and more patients are studied by *x*-ray early in the course of the disease, the number of negative films or those which show but minor changes will increase. For example, a very small stem bronchus lesion which has not totally or partially occluded the bronchus may not produce either atelectasis or localized emphysema. In these cases, the *x*-ray would be negative. Occlusion of a branch bronchus and atelectasis of only a segment of a lobe may give a very confusing shadow or one that might be missed altogether. In Figures 1 and 2 are shown reproductions of *x*-rays which illustrate this point. In the first case, atelectasis of a portion of the right lower lobe might have been easily overlooked or confused with bronchiectasis. In the second case, the distribution of the tumor in the upper lobe simulated tuberculosis. The routine use of the *x*-ray in the examination of all patients with any chest symptoms is essential. The profession should profit from the experience gained in the findings of tuberculosis cases. After many long years, it is now recognized that symptoms and physical signs in chest disease are not reliable. In order to find minimal cases of tuberculosis, people who have had contact with known cases and selective groups of the population must be surveyed by *x*-ray. In the detection of pulmonary cancer, we are not confronted with screening large numbers of apparently healthy individuals, but efforts should be centered upon that portion of the population who complain of chronic chest symptoms, notably cough.

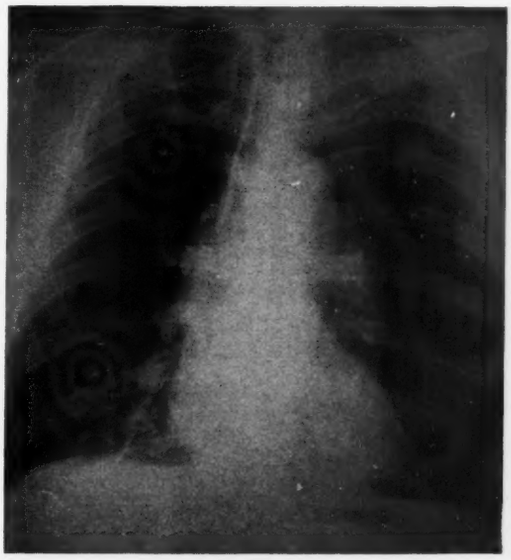

FIG. 1. Roentgenographic appearance of early stem bronchus carcinoma. Note slight increased density of lung field in right cardiophrenic region, and slight displacement of mediastinum and heart to that side. The lesion took origin in the bronchus of the right lower lobe causing only partial occlusion. Mr. A. F., age sixty-seven, was referred by Dr. J. McNamara, Taunton, Massachusetts. A chronic cough followed a chest cold seven months before admission. The sputum was at first scanty, then became more productive but was of a thin, clear, mucoid character, occasionally tinged with blood. Three months before admission the patient noticed a wheeze which appeared with increased activity. Bronchoscopic biopsy revealed an epidermoid carcinoma, Grade II. Exploratory thoracotomy was done on June 25, 1940, and tumor cells could not be demonstrated in the mediastinal glands. A right pneumonectomy was successfully performed. The patient was discharged on the twenty-sixth postoperative day after an uneventful convalescence.

THE MAJORITY OF LESIONS CAN BE SEEN AND BIOPSIED

Approximately three-fourths of all primary lung tumors which have been studied clinically have been found to be within the range of vision bronchoscopically. In Gebauer's series[2] of more than 100 bronchoscopies, the lesion was seen in 72 per cent. In a consecutive series of sixty-seven cases studied by Betts[3] a positive biopsy was obtained in 71.6 per cent of the cases. In 127 verified cases studied by the author the diagnosis was first established by bronchoscopic biopsy in 61 per cent. (Fig. 3.) Gebauer has rightly pointed out that if the time element is considered, a figure as low as 25 per cent for bronchoscopic inaccessibility is misleading as this includes a high proportion of late cases. When dealing with early lesions, perhaps 40 per cent will not be visible bronchoscopically. However,

FIG. 2. Preoperative roentgenogram and surgical specimen showing appearance of primary carcinoma involving the right upper lobe. Note that the area of density in both postero-anterior and lateral exposures does not appear as a discrete rounded shadow or as a shadow suggesting atelectasis. The patient had been under treatment for tuberculosis at one time. Mr. S. K., age fifty-five, was referred by Dr. M. J. Stone, of Boston. Illness began with an attack of pneumonia six months previously. The patient was seriously ill for seventeen days, after which there was improvement, but there was persistent cough with yellowish green sputum. Bronchoscopic examination was negative. Exploratory thoracotomy was advised and carried out on December 13, 1940. A solid mass was palpated in the upper lobe. A single infracarinal lymph gland was enlarged and upon frozen section revealed an epidermoid carcinoma, Grade III. A right total pneumonectomy and dissection of mediastinal glands were successfully performed. The patient was discharged on January 24, 1941, as greatly improved. In May, 1941, upon follow-up examination, a persistent tachycardia was found. There was marked displacement of the mediastinum and heart to the right. A thorough search for recurrent disease was negative. On May 14, 1941, a right upper thoracoplasty was performed in order to correct cardiac displacement and prevent emphysema of the left lung. The patient left the hospital sixteen days later as improved.

the number of early stem bronchus lesions discovered will be in direct proportion to the frequency with which bronchoscopic examination is carried out as a routine in all patients with bizarre chest complaints and abnormalities by *x*-ray.

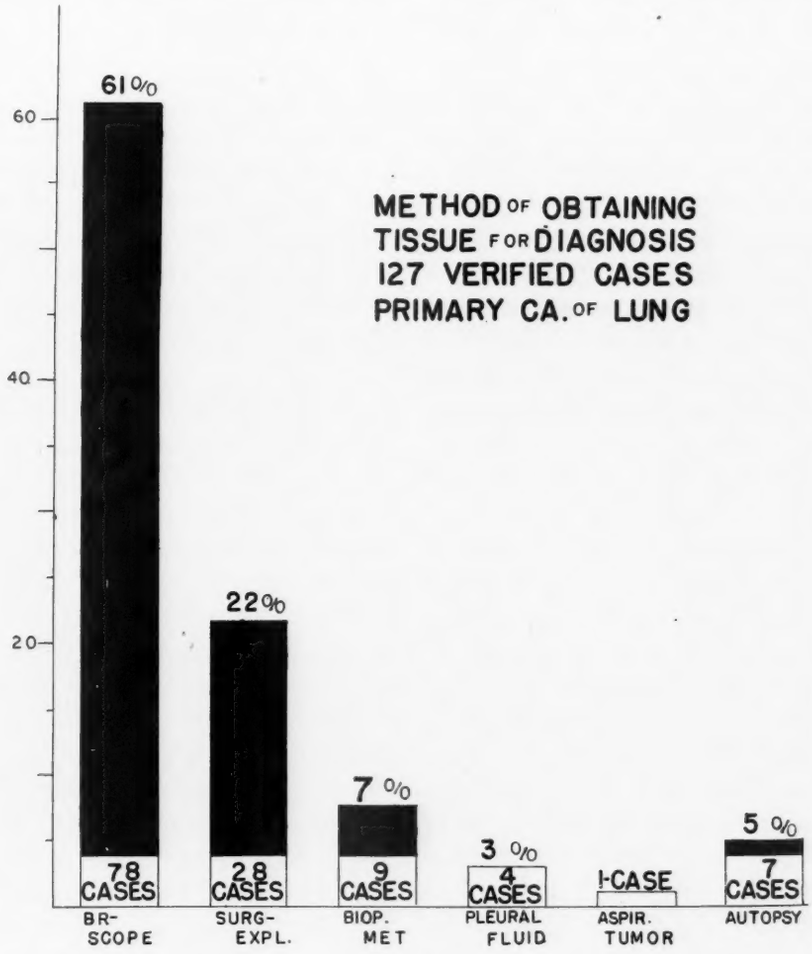

Fig. 3.

Speeding up mechanism of early discovery

An attempt has been made to show the important factors which will operate to speed up the mechanism of early discovery. A small tumor originating in the sensitive mucosa of one of the stem bronchi soon produces a chronic cough. This cough is an irritative, hacking and persistent one, which forces the patient to consult a doctor. The possibility of cancer is on the mind of the first doctor consulted. An *x*-ray is taken. If an abnormality is found such as an atelectatic shadow, enlarged hilar shadow, or opacity in the periphery of the lung field, bronchoscopy follows immediately. If the *x*-ray is negative and unexplained symptoms persist, bronchoscopy is still indicated.

The suspected case—a surgical problem

A positive bronchoscopic biopsy brings that patient up to the point of appraisal for operability and curative treatment. The patient suspected of having cancer, whose bronchoscopic examination has been negative, has been carried but half way along the path to early discovery and possible treatment. Failure to obtain a bronchoscopic biopsy should not be a cause of delay. The patient should not be permitted to slip into the hopeless group. The problem

for either the proved or suspected case at this point is still a surgical one. Many patients whose clinical investigations have been carried through this far strike a snag because opinions differ as to what the next step should be. Too often, the patient is advised to wait for absolute evidence of malignancy in order that surgery may in some way be avoided. While time is being used to wait for changes which make a diagnosis histologically positive, the patient may be robbed of his only opportunity for cure. Furthermore, many patients today with both proved and suspected malignancy are given *x*-ray therapy as the initial form of treatment. Many times, surgical treatment is not given consideration until after it has been demonstrated that the lesion has progressed in spite of radiation. Then, too often, nothing can be accomplished surgically. The criterion for the application of *x*-ray treatment should not be based on just the suspected or proved diagnosis of primary carcinoma of the lung. The true criterion for the application of *x*-ray treatment should be based on the establishment of the fact that the growth has spread beyond the lung, and is therefore, inoperable. In other words, the patient should be considered a surgical problem until inoperability has been established beyond a reasonable measure of doubt. Radiologists are almost in complete agreement that the great majority of primary bronchiogenic tumors are radioresistant. In a recent report on the present state of radiation therapy, Dresser[4] stated: "Carcinoma of the bronchus is usually not responsive to radiation although there is an occasional rare exception." Graham[5] has stated, "I have been unable to find any incontrovertible evidence that up to the present time any case of primary bronchial carcinoma has been cured by radiation therapy."

Recently, Steiner[6] studied the microscopic effect of roentgenotherapy on twenty-one patients who had been treated and all studied at autopsy. Doses up to 5,000 roentgens did not destroy the cancer as judged by microscopic standards. Survival in the twenty-one patients treated was 11.9 months as compared to 10.5 months in fifty-three nontreated patients.

It is significant that not a single radiologist reporting experiences on radiation therapy of primary lung cancer recommends its use in operable cases. At the present state of our knowledge, radiation can be used with justice only for those patients in whom inoperability has been established. Patients with suspected or proved primary carcinoma of the lung are surgical problems until proved otherwise.

APPRAISAL FOR OPERABILITY

In appraising a proved case of cancer of the lung from the standpoint of surgical treatment, there are certain evidences that should be considered as positive, others as presumptive evidences of inoperability. Quite obviously, the greatest single fact which determines operability is the presence or absence of an extension of the tumor beyond the lung. Theoretically, absolute proof of metastatic disease can be given only when metastatic tissue is examined microscopically. Frequently, it is impossible to verify metastatic disease histologically. The following clinical signs appear most frequently in evidence of inoperability: (1) Frozen mediastinum as determined bronchoscopically; enlarged mediastinal glands without signs of fixation are not always involved in a metastatic process; (2) tracheal or contralateral bronchial involvement; (3) enlargement of lymph glands (cervical glands most frequently) proved to be involved by biopsy; (4) metastasis to the contralateral lung; (5) metastatic lesions to bones on unquestionable roentgen ray evidence or biopsy; (6) engorged veins of the neck and upper chest; (7) enlarged liver or jaundice; (8) advanced age; and (9) Complicating disease which within itself will prove to be fatal.

At one time, we were of the opinion that patients should not be accepted for surgical exploration if certain presumptive signs

were present. Paralysis of the diaphragm usually means infiltration of the phrenic nerve and widespread mediastinal involvement. We have seen one such patient. Vocal cord paralysis also strongly suggests the possibility of mediastinal invasion.

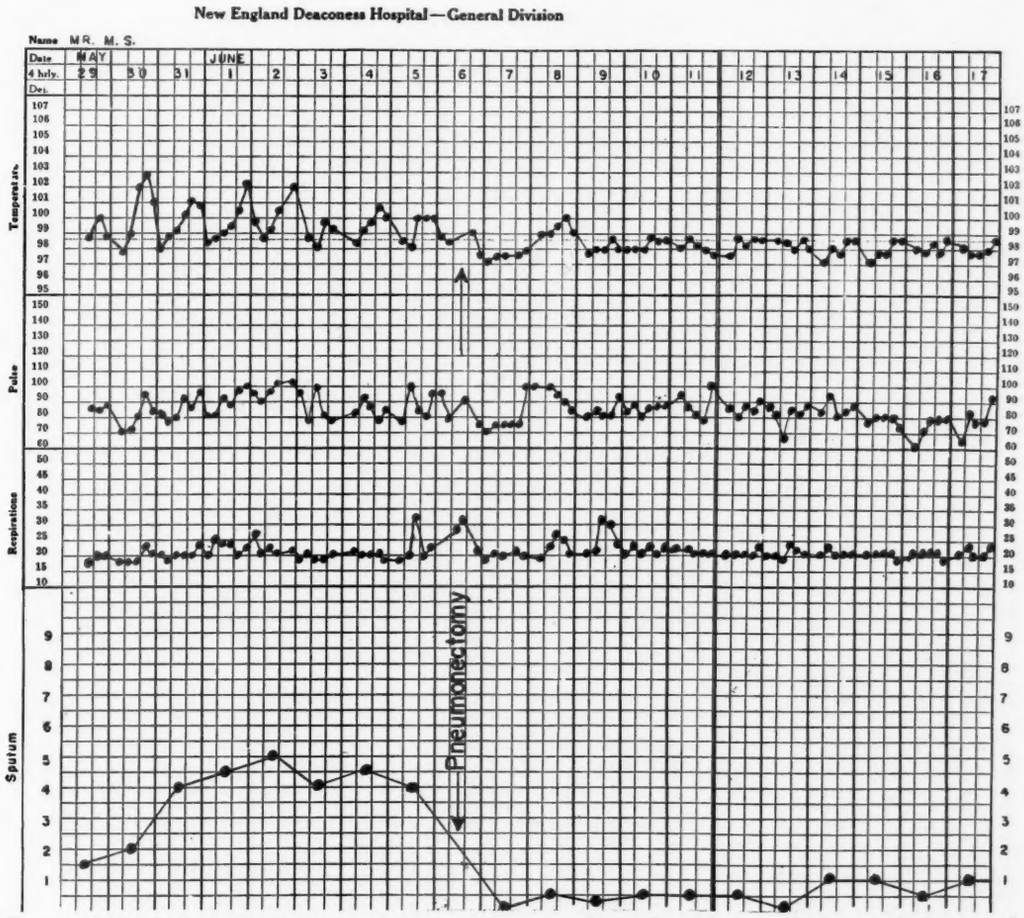

Fig. 4. Clinical chart showing record of temperature, pulse and respirations before and after total pneumonectomy. There was evidence of sepsis as well as a bronchial occluding cancer. Mr. M. S., age fifty-one, was referred by Dr. J. G. Adams of Salem, Massachusetts. A chronic cough developed four months previous to admission. The sputum was clear at first, later blood-streaked and mucopurulent. For two months the patient complained of profuse sweats, weakness and a weight loss of twenty pounds. Upon bronchoscopy an epidermoid carcinoma Grade II was found which partially occluded the left upper lobe bronchus. Considerable purulent material could be seen coming from beyond the lesion. The white blood count was 21,100. Left pneumonectomy was performed on June 6, 1941, and the patient was discharged from the hospital twenty-seven days later after a satisfactory convalescence.

ment. However, in one instance in which there was diaphragmatic paralysis, exploration was done and no mediastinal extension was found. Reservations must also be made in regard to clear pleural fluid free of tumor cells. Usually, a pleural effusion comes as a late manifestation of cancer after mediastinal lymphatics have been blocked. However, an occasional patient may have an effusion and still show upon exploration no evidence of mediastinal glandular involvement. Such a finding was encountered in three patients. All gave other evidences of inoperability and they were not explored.

Evidences of sepsis do not necessarily contraindicate pulmonary resection. Over one-half of all patients with primary carcinoma of the lung in our experience have shown effects of an associated lung infection. The toxic manifestations of a smoldering infection or suppuration distal to the occluding lesion may so prostrate the pa-

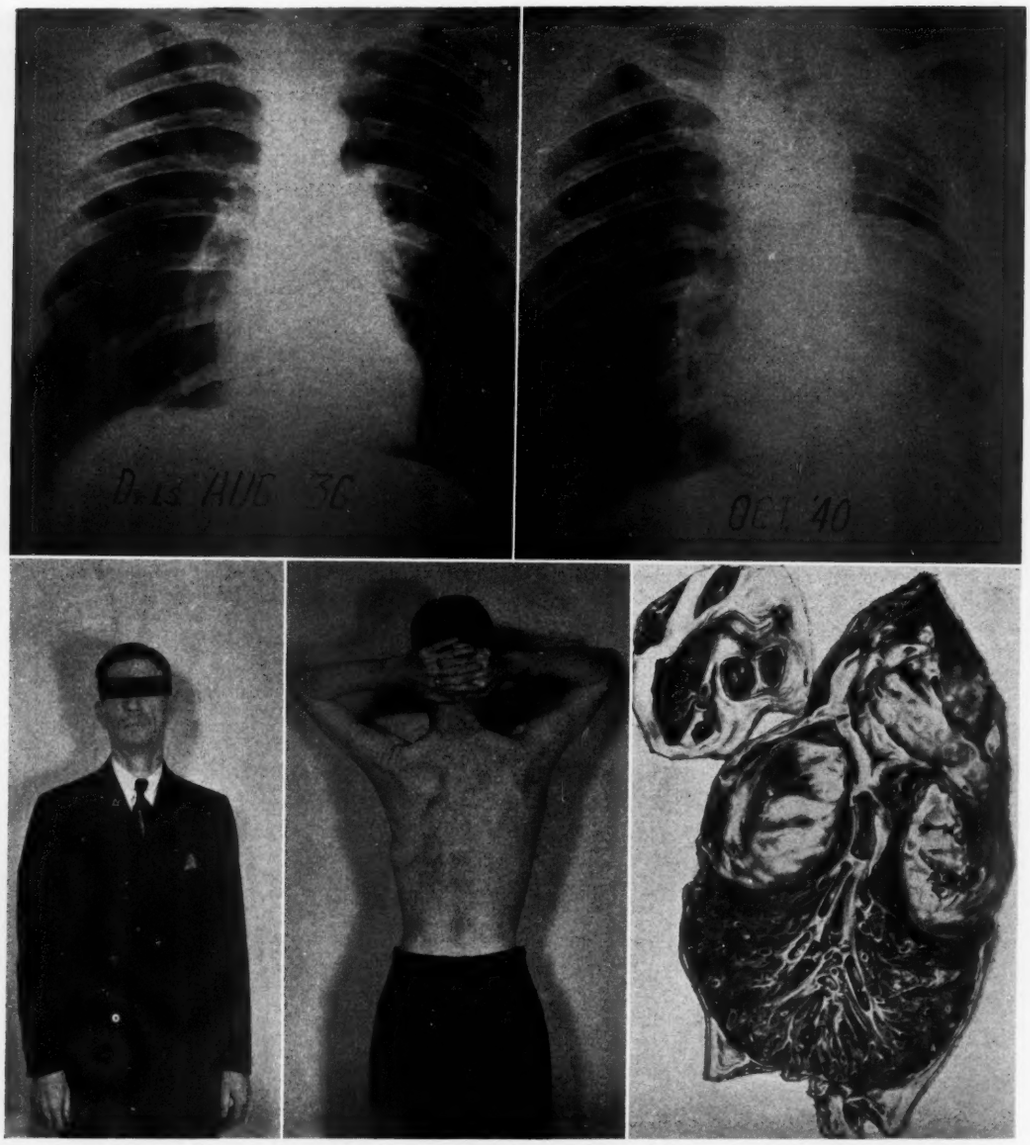

Fig. 5. Long standing disease which proved to be operable four years after onset of symptoms and five months after malignancy was first established. Note enlarged hilar shadow in 1936 film. Four years later there was a marked shift of the trachea to the left with an irregular density of the lung field. The photograph was taken six weeks after operation. Note freedom of deformity. A drawing of the sectional surgical specimen is shown. Note a well circumscribed mass which measured 6 by 4 by 4 cm. The small drawing shows the appearance of the left main stem bronchus which has been divided just above its first division in order to show the intrabronchial portion of the tumor. Dr. I. S., age forty-eight, was referred by Dr. P. G. C. Bishop, New York City. The patient had been having influenza-like episodes and attacks of bronchitis for a period of four years. The most recent attack occurred in March, 1940, with septic temperature for ten weeks and moderate amounts of purulent sputum. There was a loss of thirty-four pounds in weight. Bronchoscopy on June 7, 1940, revealed a tumor reported as malignant partially obstructing the left main bronchus. On November 1, 1941, an exploratory thoracotomy was performed. Mediastinal glands were found to be free of involvement. A left total pneumonectomy was carried out. The final pathological report was malignant infiltrating adenoma of bronchial origin. The patient was discharged forty days after operation following an uneventful convalescence.

tient that surgical intervention may be considered entirely out of the question. The procedure which removes the cancer-bearing organ will also remove the septic reservoir. In Figure 4 is shown the clinical chart of a patient suffering from a bronchial occluding carcinoma with secondary infection. Immediately following surgical extirpation of the infected and cancer-bearing lung, the patient improved and signs of sepsis disappeared.

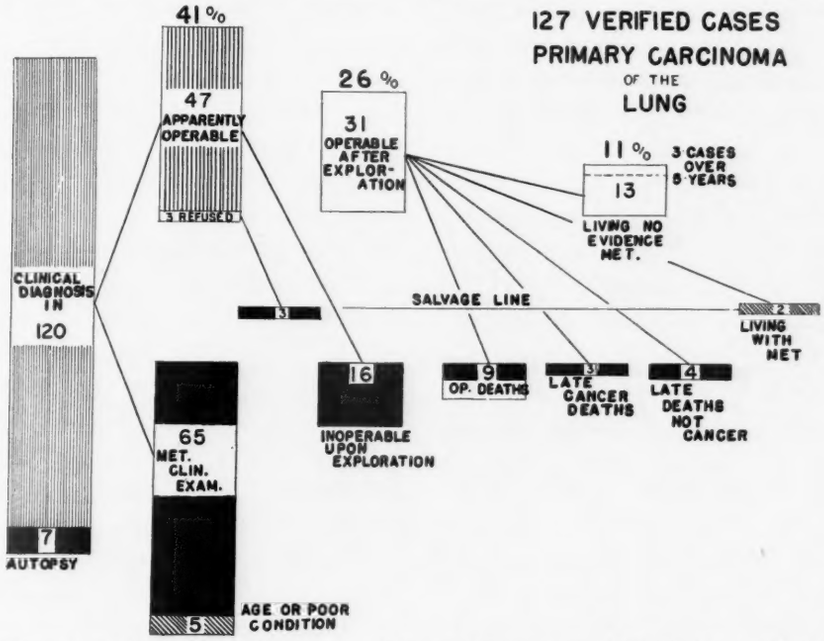

Fig. 6. A chart which shows the ultimate fate of 127 histologically verified cases of primary carcinoma of the lung. This represents all of the cases studied by the author between 1932 and March, 1941. Note the high incidence of clinical discovery. The percentage of operable cases compares favorably with operability of carcinoma of the stomach. Two of the late deaths caused by cancer occurred in patients treated by lobectomy. These cases antedated the now accepted use of pneumonectomy in the treatment of cancer. The four patients who died of other causes all received temporary benefit. It is significant that 11 per cent of the original group are living, well and free of metastatic disease.

So-called signs of pulmonary insufficiency such as dyspnea, cyanosis or tachycardia are not bona fide evidences of inoperability. Such a situation is most frequently encountered when the growth has either completely or partially occluded a primary bronchus. The flow of blood through a nonventilated lung results in the return of considerable unoxygenated blood to the general circulation. The cardiorespiratory mechanism is greatly handicapped. The effect is similar to that of a patent ductus arteriosis or a large arteriovenous aneurysm. Ligation of the pulmonary artery and extirpation of the functionless lung increases the efficiency since all of the blood passes through a sound lung.

The duration of symptoms does not necessarily bear a relationship to operability. For example, in the case of Dr. I. S., symptoms of a bronchial occluding lesion had been present for four years. A bronchoscopic biopsy done five months previously had revealed an infiltrating adenoma which had become malignant. Although the history was a long one, there was no clinical evidence of metastasis. Thoracic exploration was carried out; mediastinal glands were not involved; and resection was successful. (Fig. 5.) A long history without clinical evidence of metastatic disease may actually be a favorable sign as it strongly suggests the presence of a slow-growing tumor or low-grade malignancy.

It is significant that in the author's series of cases, of 127 verified patients there were but five who were rejected for surgery because of too advanced age or too poor general condition. (Fig. 6.) There were but fourteen cases in the group of seventy-five unverified cases who were rejected for surgery. Many of these patients were unverified because the general condition or age of the patient did not justify bronchoscopic examination or surgical exploration in order to settle the diagnosis. These ratios do indicate that from a practical standpoint very few patients need be denied the benefits of surgical treatment because their general condition does not warrant thoracic exploration.

The problem of operability is not an easy one to settle. In dealing with a disease which is 100 per cent fatal without surgical excision, we are forced into the position of giving the patient the benefit of every doubt. The evidence that the tumor has spread beyond the lung must be above reasonable doubt. This question cannot be settled fairly by anyone who has not had an opportunity to follow such patients through a surgical program.

THE PLACE OF SURGICAL EXPLORATION

The problem presented by that group of patients in whom a presumptive diagnosis was made, but unverified by bronchoscopic biopsy, warrants special consideration. Great advances have been made in the surgical treatment of tumors of the abdomen, brain, and other organs simply because surgical exploration has been freely resorted to in doubtful cases. A clearcut policy should be formulated in regard to the suspected, yet unproved case of carcinoma of the lung. Tuttle and Womach[7] have expressed an opinion and it has been our own impression that the peripherally located tumors are more rapidly growing and more likely to metastasize at an early stage than stem bronchus lesions. This may be due in large measure to the policy of watchful waiting on the part of the first doctor consulted with the hope that time would furnish additional data and in that way clear up the diagnosis. If a patient with a peripheral lesion is going to be given a reasonable chance for cure, there must be a plan for immediate attack. If one waits for the growth to enlarge so that it can be seen bronchoscopically or for additional signs, these signs will most likely present themselves as evidences of extrapulmonary extension.

Attempts to settle the diagnosis by aspiration biopsy in early cases should be discouraged. It is illogical and unwise to resort to needle aspiration in operable cases. Regardless of the outcome of the aspiration, surgical exploration should be done. The positive case requires exploration for curative treatment. The negative case requires exploration for establishment of diagnosis and treatment. There remains a small minority with peripheral tumors showing evidence of extrapulmonary involvement in which aspiration biopsy may be used. In certain cases, however, a biopsy obtained through a small thoracotomy incision may be employed to double advantage. The open biopsy can be done with extreme safety and a larger specimen will prove to be more reliable from the standpoint of cellular arrangement and architecture.

The policy of recommending thoracic exploration in suspected cases of primary malignancy of the lung is sound and is the safest course to follow. For example, we found thirteen operable malignant lesions in a series of thirty suspected cases who were explored. (Fig. 7.) Nine of these patients were salvaged. Three have since died, leaving six who are living and free of evidences of metastatic disease. A watchful waiting plan would have meant death for all thirty. Prompt surgical exploration in future groups in whom the disease is suspected at an earlier stage by the first doctor consulted will raise the salvage rate considerably.

In addition to what one may do to prevent a death from cancer, there are other benefits in exploring doubtful cases.

If the lesion is not due to a cancer, there is a strong possibility that appropriate surgical treatment can be given. For example, it is at times impossible to differentiate clinically between a pulmonary abscess and a tumor. This is especially true when the onset has been insidious, when the lesion is fairly well circumscribed, and a fluid level is not seen by *x*-ray. One may proceed with a small thoracotomy opening which can be converted into a drainage tract should the lesion prove to be simply an abscess. Again, it is at times difficult to distinguish a small localized interlobar empyema from a tumor. Surgical treatment is indicated for either condition. For example, a small interlobar collection of purulent material of long-standing caused cough and hemoptysis in two of our cases. There was no elevation of temperature or white count. Bronchoscopy was negative and there was a lack of bronchial filling of the area after lipiodal. A tentative diagnosis of primary carcinoma of the bronchus was made and surgical exploration advised. In both cases, malignancy was ruled out and drainage of the areas resulted in cure. Three other extremely ill patients were thought to have primary malignancy but were suffering from multiple abscess of the lung. In two of these patients, both the upper and lower lobes were involved and pneumonectomy was successfully performed and was curative. In a third case, Miss B. L., a mass three inches in diameter was found in the left lower lobe which, after lobectomy, proved to be a chronic multilocular abscess. These cases are cited to illustrate the importance of attacking questionable cases in a straightforward surgical way. The diagnosis can be settled and curative treatment can be instituted.

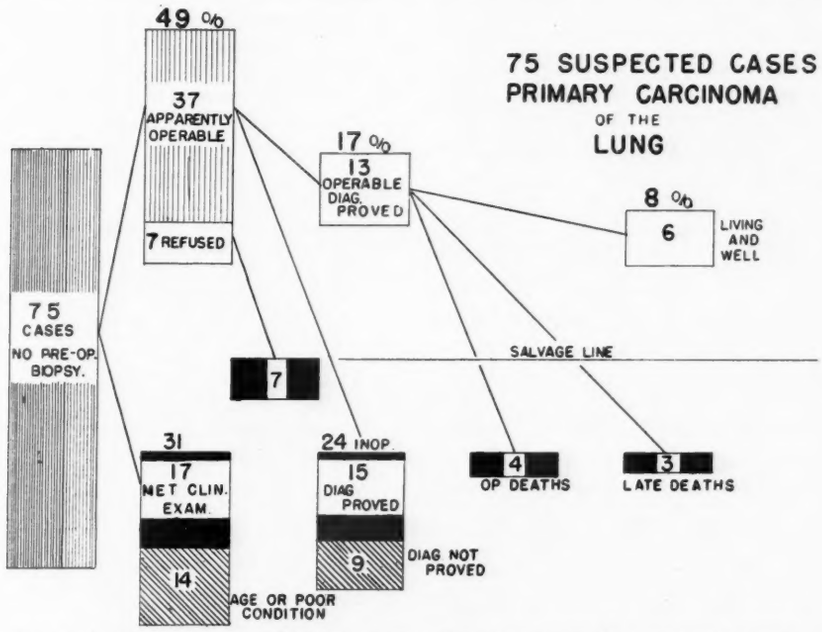

Fig. 7. A chart is shown which illustrates the salvage rate in a series of seventy-five patients suspected of having primary carcinoma of the lung. Of this group almost one-half showed no clinical evidence of an extension of the growth beyond the lung and surgical exploration was done. It is significant that of those cases explored 35 per cent proved to be operable. Twenty-eight of the above cases are also included in the chart shown in Figure 6, as the diagnosis was verified histologically after exploration.

CONCLUSIONS

Carcinoma of the lung has become of vital concern to every practicing physician for four good reasons: (1) Malignancy originating in the lung is near the top of the list as a cause of cancer deaths. (2) It is curable in its early stages. (3) Methods are

available for detecting early cases. (4) There is reason to believe that the majority of patients consult a doctor at a time when the lesion is still in an operable stage.

Emphasis has been placed upon practical measures by which all doctors can contribute in the control of this disease: (1) Methods which will aid in speeding up the mechanism of early discovery have been outlined. (2) A plea has been made for an agreement on management for all suspected or proved cases with special reference to radiation versus surgery. (3) Criteria of inoperability have been discussed. (4) In patients presenting presumptive evidence of cancer without bronchoscopic visualization, the value of surgical exploration has been emphasized.

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When one is confronted by a simple colloid goiter of some age the possibilities may exist that it is doing one of three things: nothing: becoming toxic; or degenerating. When the goiter is doing nothing it speaks the language of a small boy; it has not yet done anything that can be discovered. One may be pretty sure a colloid goiter is already mischief bent.

DIFFERENTIAL DIAGNOSIS OF PRIMARY AND METASTATIC MALIGNANCY OF THE LUNG*

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PRIMARY carcinoma of the lung has been brought into the fold of curable cancers. More and more frequently the problem of differentiating growths which have originated in the lung from a "solitary" pulmonary metastasis must be solved.

Primary tumors of the lung can be conveniently separated as to location into central and peripheral groups. The former have their origin in one of the larger bronchi. These lesions can be seen and biopsied bronchoscopically. Tumors of the central group soon occlude a large bronchus with resulting atelectasis, which may be suspected by physical examination and will be detected roentgenologically. Differential diagnosis is not difficult. Peripheral tumors arise in smaller bronchi and, therefore, are not visible at bronchoscopy. Peripheral tumors do not occlude large bronchi in the early stages so that signs of atelectasis are lacking. Unless the tumor is large they produce no abnormal physical signs. On roentgenological examination they usually present themselves as more or less rounded discrete masses, but many times the shadow may be confused with other conditions.

The differential diagnosis of peripheral lesions is too often deferred until the growth produces marked symptoms and gross disturbances with involvement of pleura or distant metastasis. If we wait until the gross changes are unmistakable, we shall make no mistakes in diagnosis, perform absolutely no unnecessary operations and cure practically no patients. Our diagnostic accuracy and ultimate mortality will be close to 100 per cent.

It is the purpose of this paper to discuss the differential points of the peripheral primary lung cancer and solitary metastasis from an extra pulmonary primary lesion.

THE LUNG AS A FILTER FOR TUMOR METASTASIS

The lung is a mirror of the human body for cancer. Malignant growths, when considered collectively, cause death by pulmonary metastasis in 30 per cent. In growths originating in organs not drained by the portal system, the incident is even higher, 36 per cent. Thus, by including the 10 per cent of deaths which are due to primary carcinoma of the lung, we obtain a view of 40 to 46 per cent of all the cancers of the body by looking into this mirror.

It should be made clear that involvement of tissues by contiguity, either from primary or secondary growths, is not metastasis. Only emboli which have arisen from the primary growth as detached, transported fragments of that tumor are entitled to be called metastasis. Pulmonary metastasis may be solitary but is usually multiple and bilateral. In the latter case, the patient is obviously beyond the range of surgical aid.

The differential diagnosis is concerned primarily between the primary peripheral and the solitary pulmonary growths. There is evidence from postmortem examination that solitary metastasis occurs in some instances. Lenke[1] reports three cases with a single deposit in the lung; two were from a hypernephroma of the kidney; the other was a chorion epithelioma. Brandt, Pirchan and Sikl[2] have reported cases of renal carcinoma with a single

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deposit in the lung as the only metastasis. Barney and Churchill[3] reported the successful removal of a "solitary" pulmonary metastasis from a primary kidney tumor. The patient was alive and well after five years.

In cases such as these, are we to assume that a solitary tumor embolus was discharged from the primary growth and that it finally reached a fortuitous destination? Or are we to assume rather that multiple emboli were discharged from the primary growth but that only one of these survived to grow in some favorable situation? Tumor embolism is not metastasis. Many neoplastic emboli perish or remain sterile in the new sites of arrest. The "seed" has failed to germinate because the "soil" is poor.

Tumor emboli to the lung arise from tumor invasion of the systemic veins and metastatic growths in the lungs arise from these malignant emboli carried in the pulmonary arteries to be arrested in the smaller vessels. The first minute vessels encountered by emboli liberated from the other organs are the pulmonary arterioles and capillaries; and most systemic venous emboli suffer arrest in this great filter. Emboli apparently arise in periodic showers. In some cases multiple growths are all of similar size, suggesting a simultaneous origin in one shower of emboli; in other cases growths of all sizes may be present, indicating embolic inoculations of different ages.

CLINICAL CONSIDERATIONS

The diagnosis of either primary peripheral or "solitary" metastasis of the lung must be based on the summation of all the clinical data. A careful search should be made for a possible primary site elsewhere. Complete kidney and gastrointestinal roentgenological studies should be made. The absence of a positive extrapulmonary primary site throws the weight of evidence in favor of a primary pulmonary tumor. Although absolute differentiation between the two lesions is many times impossible, certain clues, which may be of help, are summarized in the table below:

DIFFERENTIATION OF PRIMARY PERIPHERAL AND METASTATIC PULMONARY CARCINOMA

Primary Peripheral Carcinoma	Common to Both	Metastatic Carcinoma
Symptoms		
Hemoptysis is fairly common.	There may be none whatever. Pain or discomfort of the chest may be present due to peripheral or subpleural location. There is little difference in the frequency or character of cough.	Hemoptysis is uncommon.
Signs		
	There may be none. These are variable depending on size and location of tumor.	
***X*-ray**		
The lesion is single...	.....	The lesion is usually multiple.
The lesion is unilateral.	.....	The lesion is usually bilateral.
The lesion is more often in the upper lung fields.	.....	The lesion is apt to be in the lower lung fields.
Cavitation is more frequent.	.....	Cavitation is infrequent.
Bronchoscopy		
	Both types are usually beyond bronchoscopic range.	

Methods other than bronchoscopy for obtaining tumor cells for verification of diagnosis are of little practical value in operative lesions.

A study of the cytology of pleural fluid when present is important in obtaining a diagnosis. It is a late sign, however, and evidence of inoperability.

We have examined the sputum carefully in our cases and have occasionally recovered tumor cells in far advanced cases. We have not been able to determine accurately the type of tumor from such studies.

Transpleural aspiration is highly recommended by Craver.[4] This method is useful only in cases which are obviously inoper-

able. To the thoracic surgeon it is a useless procedure for if the biopsy is positive in a suspected case without clinical evidence of metastasis, exploratory thoracotomy is indicated; and if the biopsy is negative in the same type of case, the same exploration is still the next step in its management.

The following case reports, illustrating many of the points described in regard to differentiating between primary and secondary malignancy, are printed in detail. Both patients are alive and well today because the problem of differential diagnosis was settled by exploratory thoracotomy.

The following case history relates the course of a patient who had a resection of the cecum for adenocarcinoma. She remained well for four years. Then she developed respiratory symptoms and was found to have a shadow in the chest film suspicious of malignancy. This patient is at the present time enjoying good health without evidence of disease, three years after pneumonectomy.

Case 1. Miss S. S., a fifty-three year old, single, Syrian woman, was referred by Dr. Jacob Auslander of New York City. She was admitted to the New England Deaconess Hospital on the service of Dr. Richard H. Overholt on April 8, 1938, with a chief complaint of cough and hemoptysis of one year's duration. The one significant fact in the past history was that the cecum had been resected for neoplasm four years previously by Dr. A. A. Berg.

The patient stated that she had felt well and continued to work until one year prior to admission. Quite insidiously, in the spring of 1937, she began to raise small amounts of blood, associated with a slight cough. Several times a week this event was repeated. Shortly thereafter, a vague sense of discomfort in the left lower anterior portion of her chest was noted. She went to a doctor and a chest *x*-ray was taken which disclosed an area of increased density in the left lower lung field. The patient continued coughing and raising small amounts of blood without the development of additional symptoms. Concern about the continued hemoptysis led her to seek further investigation and Dr. Auslander was consulted.

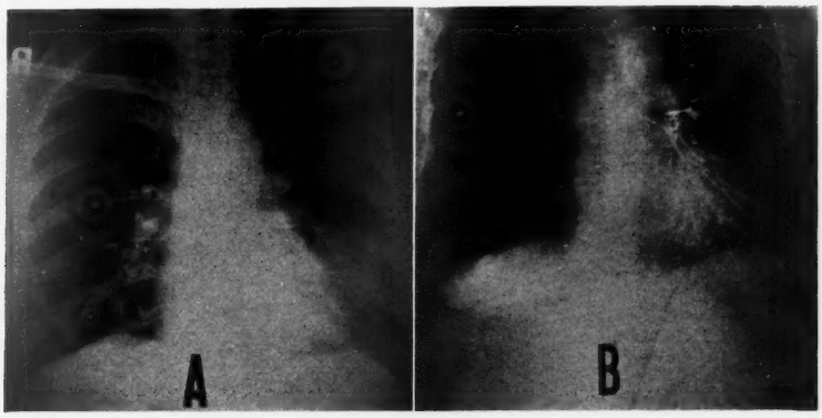

Fig. 1. Case 1. Postero-anterior *x*-rays of the chest are shown. A, shows diffuse haziness in lower left lung field; B, bronchogram revealing blockage of lingular branch of the left upper lobe.

Physical examination revealed a well developed, obese woman of fifty-three years. The only positive findings were diminished breath sounds over the midaxillary area of the left chest. A well healed, right rectus scar was present. Rectal and pelvic examinations showed no abnormality.

Roentgenographic examination of the chest revealed an area of increased density in the anterior portion of the left lower lung field. (Fig. 1.)

Bronchoscopic inspection of the trachea and major bronchi showed no abnormality. A very good visualization was obtained of the left upper lobe bronchus, it being possible to see up the bronchus as far as a spur between its next division, but no evidence of new growth or other abnormality was noted. Under fluoroscopic control, bronchographic studies of the left bronchi were made. An occlusion of the lingular branch of the left upper lobe bronchus

about 1 cm. distal to its origin from the left upper lobe bronchus was found.

Skeletal roentgenograms were negative for evidence of metastatic disease. No pathological condition of the kidney was revealed by pyelogram. A barium enema showed no evidence of local recurrence. The pathological report of the previously resected cecum was secured. It reported adenocarcinoma of the cecum without evidence of lymph-node metastases. Although the probability was held that the nodule in the left lung was a late metastasis from the cecum, it was impossible to rule out a peripheral primary carcinoma; and due to the fact that serial *x*-rays showed that a definite increase in its size had taken place in the last four months, an exploratory thoracotomy was thought advisable.

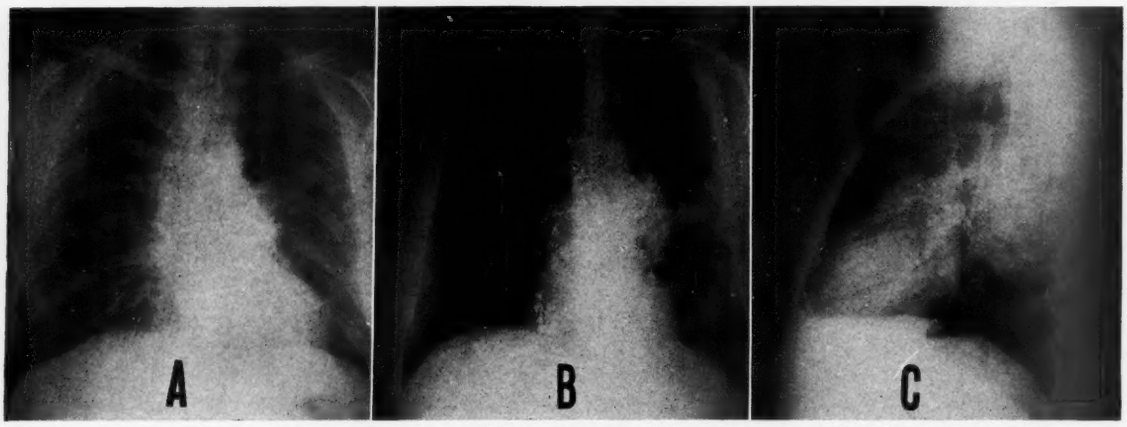

FIG. 2. Case II. A and B, postero-anterior and left anterior oblique *x*-rays of the chest show the opaque, irregular shadow at the left hilum; C, left lateral *x*-ray reveals the suspected tumor to be posterior.

Pneumothorax was then instituted on the left side and maintained for three weeks. On April 11, 1938, an exploratory thoracotomy was performed under cyclopropane intratracheal anesthesia. The tumor presented itself in the lower part of the upper lobe as a firm well circumscribed mass which puckered the lung tissue and was about one and one-half inches in diameter. The parietal pleura was not involved. A frozen section of a gland from the hilum showed no evidence of malignancy. Pneumonectomy was performed, with individual dissection and ligation of the hilar structures.

The pathological report of the tumor removed from the lung was as follows:

"Left lung—weight 330 gms. Lower portion of upper lobe contains a firm nodule 6 cm. in greatest diameter with a puckered area 4 cm. in diameter on surface. Cut surface of nodule is mottled gray, yellow, red and black. Bronchi and vessels appear negative at hilus. Microscopic diagnosis: Adenocarcinoma, probably metastatic from gastrointestinal tract."

The patient's postoperative course was uneventful and she was discharged from the hospital on May 10, 1938, twenty-one days after operation. The patient was last examined on May 16, 1941, three years after pneumonectomy. She reported that she had been enjoying good health. There were no symptoms referable to the chest or abdomen. She was employed as a saleslady. Physical examination revealed no signs of metastatic disease.

CASE II. Mr. N. W., a fifty-five year old, retired, widowed business man was referred by Dr. Charles W. McClure of Boston. He entered the New England Deaconess Hospital on January 23, 1941, with a chief complaint of nausea, vomiting and anorexia of one year's duration. Family history and past history were negative.

The present illness dated to the sudden death of his wife one year previously. Following this, he noted anorexia and later nausea and vomiting. There had been frequent and excessive use of alcoholic beverages. After obtaining no relief from various medicines, a gastrointestinal roentgenographic examination was advised. While these *x*-ray studies were being carried out, an abnormality in the left lung was noted. Chest symptoms were not present except for a slight cough of fifteen years' duration, attributed to cigarettes. Although the patient had no urinary symptoms, he had been informed one week previously by his physician that blood was found in the urine.

Physical examination revealed a well developed and well nourished man with evidence of slight weight loss. A few crackling rales were noted in the left mid chest posteriorly, and transient moist rales at the right base which cleared after coughing. The heart was within normal limits; the blood pressure was 160/88. The remainder of the physical examination was entirely negative.

Laboratory examinations revealed the red blood count to be 3,540,000; hemoglobin, 68 per cent; white blood count, 10,300; nonprotein nitrogen 30 mg. per cent. The blood Hinton was negative. The urine was grossly bloody and the sediment was loaded with red blood cells.

Complete urinary studies were made by Dr. Harvard Crabtree, and ureteral catheterization showed the blood to be coming from both kidneys. Retrograde pyelograms revealed no etiological factor. The urological consultant suggested a diagnosis of idiopathic hematuria.

X-ray examination of the chest showed an infiltrative lesion extending out in the region of the left hilum suggestive of neoplasm. (Fig. 2.)

Bronchoscopic examination was negative except for a slightly granular appearance of the left main stem bronchus at the level of the upper lobe orifice. A biopsy showed no evidence of malignancy.

After a preliminary left pneumothorax was established, the chest was explored on February 13, 1941. A tumor mass about the size of a lemon was encountered in the left lower lobe. Several lymph-nodes were removed which were negative on frozen section. A one stage left pneumonectomy was carried out. The pathological report was as follows:

"Specimen consists of four soft, dark gray nodes. Also a left lung weighing 370 grams. The upper lobe is partially subdivided into two lobes. The pleural surface is mottled light pink and dark gray. The substance of the lung is crepitant throughout except for an intrinsic mass roughly 6 cm. in diameter which involves the apex of the lower lobe and hilus and is firmly bound to the main bronchus. There are several large, only slightly firm anthracotic nodes in the hilus. Microscopic diagnosis: Epidermoid carcinoma, Grade 1. Five lymph nodes negative."

The patient's convalescence was quite satisfactory although he developed a transient auricular fibrillation on the third postoperative day. He was discharged on January 31, 1941, forty-five days after operation. When last seen on June 27, 1941, he was feeling perfectly well and was asymptomatic. Evidence of metastatic disease could not be found.

DISCUSSION

The chest *x*-rays in both these cases lead one to suspect carcinoma strongly, but a verified diagnosis could not be made. How shall we handle these cases in which we have presumptive evidence of malignant disease of the lung, but in which the clinical evidence is not conclusive? Shall we temporize and await developments, trusting that the condition is not malignant? Shall we irradiate prophylactically, or shall we explore the suspicious lung? If we temporise, then we have made Time the executioner again rather than the great healer. We believe that preoperative irradiation is not advisable in those cases in which the diagnosis is uncertain and in which there is demonstrable evidence that extrapulmonary extension of a possible malignant process has taken place. In these cases one wishes to see things as they are. The suspected early tumor is probably small. If it is also liquefied or partly destroyed by irradiation, the tumor becomes much more difficult to recognize. The concurrent changes in the surrounding tissue then present a very confusing picture. Tissue cannot be counted on to heal properly when its vascularity has been greatly impaired by radiation.

It therefore seems best that when the evidence points to the probable diagnosis of lung tumor, even though it is not conclusive, the proper treatment is immediate exploration of the suspected lung, just as an exploratory laparotomy is indicated in a suspicious lesion in the stomach or bowel when the presence of malignancy cannot be excluded. One might say that it is unscientific and unjustified to explore all cases of suspected peripheral tumor; yet it is even more unfortunate to withhold indicated treatment if the patient has an early

primary lesion. "Solitary" pulmonary metastasis does occur and may be indistinguishable from a primary growth. If the primary malignancy has been removed and a solitary lung lesion is the only sign of metastatic disease, resection of the lung and metastatic nodule appears to be justifiable.

CONCLUSIONS

Difficulties arise in differentiating primary peripheral cancers of the lung from solitary metastatic lesions. Both lesions are beyond the range of the bronchoscope. If an exhaustive search for a primary extrapulmonary tumor is negative, evidence will weigh heavily in favor of primary lung cancer. These patients should receive the benefit of an exploratory thoracotomy.

If a suspicious solitary lung lesion is discovered at a reasonable interval of time after removal of a primary tumor elsewhere and the lung findings present the only evidence of a possible metastatic lesion, exploratory thoracotomy is indicated.

Cases illustrating these two principles have been cited. In both cases exploratory thoracotomy settled a doubtful diagnosis and led to treatment that promises to be curative.

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BRONCHIAL ADENOMA*

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THE necessity for separating bronchial adenoma from other tumors of the lung has existed since 1932[1] when they first became clinically recognizable. Accurate diagnosis goes hand in hand with development of adequate therapy. The treatment of bronchial adenoma is different from the treatment of cancer of the lung, or tuberculosis, or bronchiectasis, with which adenoma is frequently confused. Treatment must be based upon sound knowledge of the life history of the tumor as well as an accurate diagnosis. The accurate diagnosis of bronchial adenoma should include more than the discovery of its mere presence. It ought to indicate the exact location with reference to the lobar bronchi, the presence or absence of bronchial obstruction, the size, shape and extent of the extrabronchial tumor, the nature of the endobronchial portion, its relations to the inner bronchial wall, and, finally, the condition of the distal lung. To obtain this information in any patient with a polypoid bronchial tumor, a broad knowledge of all the features of bronchial adenoma is essential.

It is the purpose of this paper to set forth a concise description of bronchial adenoma in all its phases.

Our concepts are based upon a 100 per cent follow-up of nineteen cases (some of which have a clinical course of twenty to twenty-five years) from the Thoracic Surgery Clinic at the University of California Hospital, and an additional forty cases personally reviewed in various clinics of this country. Probably all the protean manifestations and the natural history of bronchial adenoma have not been covered in the study of this group of cases. Newer knowledge may change our future concepts. However, when we undertook this study we had no preconceived ideas or hypotheses. Our conclusions were forced upon us from the careful study of the cases including a correlation of their history, diagnosis, treatment and pathological findings.[2–6]

The history and evolution of the separation of bronchial adenoma from carcinoma may be divided into three periods: The first began in 1882 with the publication of Mueller's case,[7] and ended in 1932 with the report on bronchial adenoma by Wessler and Rabin.[1] This period may be described as the postmortem recognition of bronchial adenoma.[8–14] The second period, from 1932 until 1938, may be called the period of clinical recognition and treatment. During this period, several important papers appeared.[15–19] Since 1938 until the present time may be called the period of development of rational therapy based upon new knowledge of the natural and therapeutic life histories of these tumors.

As one reviews the literature, he is struck by the confusion, particularly regarding the terminology applied to what is apparently the same tumor which we now term bronchial adenoma. Ephraim,[20] in 1911, termed these tumors sarcoma of alveolar pattern; Kreglinger[10] (1913), cylindrical cell carcinoma; Geipel[14] (1931), basal cell cancer; Wessler[1] (1932), benign bronchial adenomata; Kernan[15] (1935), carcinoids; Moersch[21] (1935), adenocarcinomas; Clerf and Crawford[22] (1936), benign glandular tumors; Zamora and Schuster[23] (1937), vascular adenoma; Welt and Weinstein[24] (1937), endotheliomas, and finally Womack and Graham[17] (1938), mixed tumors of the lung.

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DURATION AND SEX

The age of the patient at the onset of symptoms of adenoma is strikingly different from that of patients with carcinoma. In our nineteen cases, twelve or 63 per cent had symptoms before the age of forty in contrast to only 11 per cent in Brunn's large series of carcinomas.[25]

The distribution by sex is likewise in marked contrast to that of carcinoma. In our nineteen cases of adenoma, twelve or 63 per cent were females, whereas only 9 per cent of 111 cases of our carcinomas were females.

In prognosis and duration of life, also, patients with adenoma present a marked contrast to those with carcinoma. If all cases of probable adenoma which have been reported as carcinoma are excluded, only a small percentage of "five year cures" of bronchial carcinoma remain. Notable among these is Graham's first case, now in its ninth year.[26] On the other hand, the prognosis for patients with adenomas is very good, and many "five year cures" have been reported. In our series, for example, all but three, or sixteen patients (84 per cent) lived more than three years after the onset of symptoms, fourteen (74 per cent) more than five years and seven (37 per cent) more than ten years. These figures are noteworthy since in Brunn's series[25] of carcinomas only 4 per cent of 297 patients survived three years or longer.

PATHOGENESIS

The pathogenesis of all polypoid bronchial tumors leaves much to be desired. One can make few statements that can be proved or that are founded on fact. Many authors have given names to these tumors which have suggested their origin from a specific cell or tissue. It has become fashionable these days to assume the origin of cancer of the lung and, in fact, of all epithelial bronchial tumors to be from the reserve cell found in the bronchial wall. According to this view then, bronchial adenoma should arise from the reserve cell. If we accept Womack's and Graham's interpretations,[17] we must postulate that bronchial epithelial tumors have an origin in more than one type of cell from vestigial fetal lung buds; and so this mixed origin gives rise to the production of mixed tissues seen in the adult tumor where bone and cartilage, along with epithelial patterns, are not uncommon. If we accept the work of Allen,[27] it would not be necessary to assume the origin from mixed fetal tissues, because he says that adult epithelial cells may differentiate into cartilage or bone under stimuli of repeated trauma, infections and local changes within the tumors. Kramer and Som[28] have called attention to this process of differentiation and believe it occurs in bronchial adenomas. According to Womack and Graham's concept,[17] adenomas should be regarded as potentially malignant, and the discovery of rare mediastinal lymph-node metastases is evidence in favor of this viewpoint. According to Bremer,[29] bronchial adenoma represents residual fetal lung. Welt and Weinstein,[24] and Tudor Edwards,[30] argue for endothelial origin. Reissner,[12] and Wessler and Rabin[1] believe that tumors arise from the bronchial mucous glands or from their ducts. Our own concepts are that bronchial adenomas arise from the mucous glands and their ducts which, being mixed glands, are apt to have varying patterns, and which in general follow the behavior of adenomatous tumors arising from these glands elsewhere in the body. Bone and cartilage formation is probably the result of de-differentiation of adult tumor cells.

Regardless of the tissue of origin, the high incidence in women and the occurrence of pulmonary hemorrhage with the menstrual bleeding suggests that circulating hormones may bear some relation to the pathogenesis of these tumors; but this is highly conjectural.

GROSS PATHOLOGY

Bronchial adenoma is almost always connected with a large bronchus, usually has an endobronchial portion obstructing this

bronchus and, in approximately 90 per cent of cases, has an extrabronchial extension (Fig. 1A and B) frequently larger than capsule subdivide the tumor and give to the peripheral surface a bosselated appearance, while the cut surface thus receives a

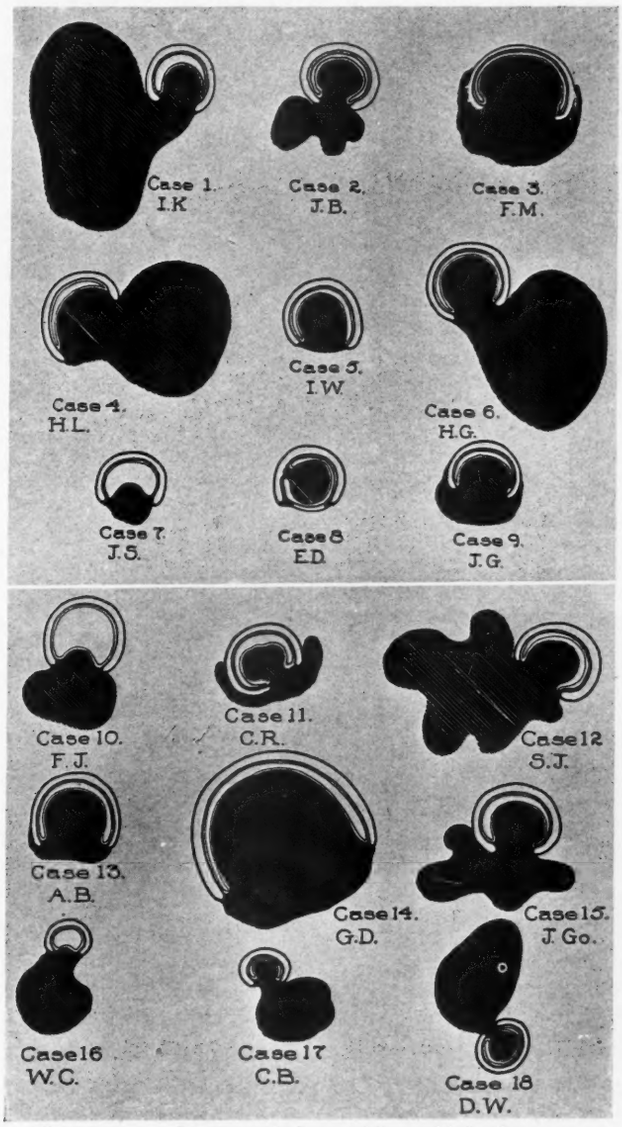


FIG. 1. A. and B, morphology of bronchial adenoma.

the endobronchial portion. The endobronchial portion is covered by mucosa which may undergo a complete metaplasia into squamous cell type (Fig. 2) and may have branches of the bronchial vessels coursing over its surface. Beneath this mucosa, whether metaplasia has taken place or not, is a layer of fibrous tissue, usually dense hyaline connective tissue but sometimes a loose, delicate, many-layered fibrous tissue. Extensions from this fibrous papillary pattern. When bone is present throughout the tumor, the otherwise smooth surface becomes rough and the projecting spicules are readily felt by the palpating finger. The extrabronchial portions projecting into the mediastinum or surrounding lung are also encapsulated, but of course their capsule is not covered with mucosa. At times a tremendous endobronchial growth of the tumors thins the bronchial rings and causes them to be dis-

placed and surrounded by tumor. Although the bronchial cartilage is not invaded, it may become atrophic. Because of the atrophy and distention of the bronchial wall, it is often difficult to determine whether all the tumor is within the bronchus or has pressed its way out into the lung or mediastinum. The location in a distended large bronchus causes the pulmonary artery and veins to be stretched over the tumors and the usual hilar relations become somewhat distorted. Longstanding pulmonary suppuration enlarges the mediastinal lymph-nodes but they contain no tumor in a careful study of our cases.*

* In one patient operated upon by Dr. Edwin Churchill and demonstrated by Dr. Castleman, there was contiguous extension into one mediastinal lymph-node; and a second similar case was recently shown us by Dr. David Wood.

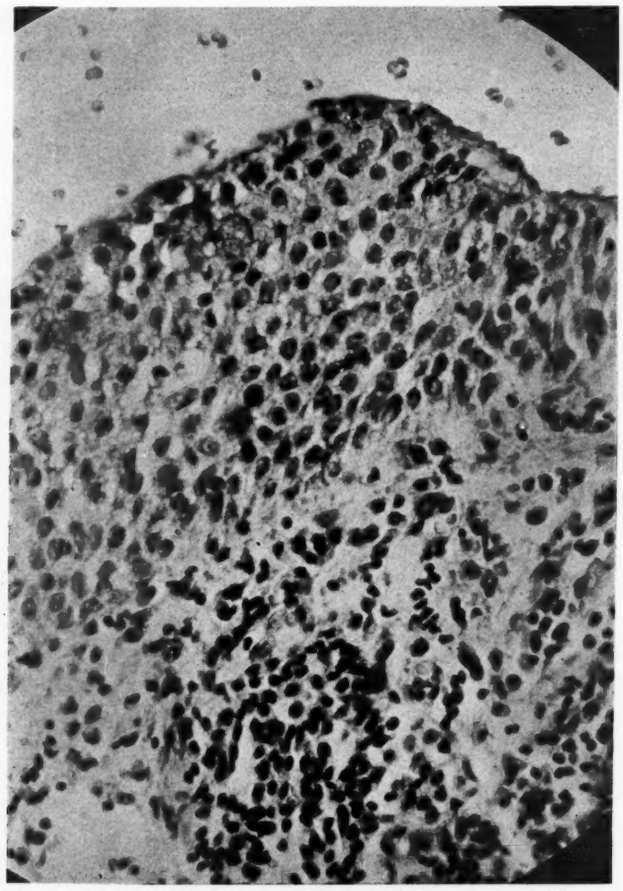


FIG. 2. Case VI. H. G. Bronchoscopic biopsy, 1933. Shows metaplasia of the mucosa overlying a tumor. Note the pyknotic nuclei and the indefinite patterns of the small tumor cells underlying the mucosa. × 120.

HISTOPATHOLOGY

The cell type (Figs. 2 and 3) which characterizes bronchial adenoma is a small cuboidal or polygonal cell, which is uniform, with an oval or round, darkly staining nucleus. The chromatin is finely divided and there are no large nuclei. Mitotic figures are absent. There is a tendency for the cells to be grouped and to form several types of patterns. These may be distinguished as alveolar, columnar, medullary, acinar, mosaic and angiomatoid patterns. A delicate reticular stroma runs through the tumor, encompasses the groups of cells and is contiguous with the fibrous tissue capsule. At times the stroma surrounds large vascular spaces. Particularly at the periphery of the endobronchial portion of the tumor, vascularity is apt to be prominent. In some cases the histologic

pattern is uniform throughout the whole tumor but in others the patterns vary a great deal in different parts of the tumor. The stroma may vary from a well developed, reticular, delicate network to a thickened or hyalinized framework which may contain large vascular spaces. In the fibrous tissue capsules, groups of tumor cells may be seen. Bone and cartilage are present in some tumors. (Fig. 3F.)

Microscopic diagnosis of adenoma from the material removed at bronchoscopic biopsy is not always easy unless the tissue shows characteristic patterns. Squamous cell carcinoma, because of squamous cell metaplasia present on the mucosal surface, angioma or angio-endothelioma because of the vascularity at the periphery of the tumor, inflammatory tissue when the epithelial surface has been altered by infection or trauma, small cell carcinoma when the biopsy has been crushed or the cells seen are not arranged in characteristic patterns—any of these may be diagnosed instead of adenoma. However, once typical patterns are obtained the diagnosis is easy to a pathologist familiar with these tumors.

COMPLICATIONS

Complications of these bronchial tumors are important since they frequently bring about the entire presenting clinical picture, and one is apt to discover patients being treated for pneumonia, bronchiectasis, lung abscess or empyema when adenoma is the hidden primary condition. If the bronchial obstruction is located in the stem bronchus, complete atelectasis will result sooner or later with infection in all the lobes of the obstructed lung. Anaerobic infection produces virulent, putrid abscesses, but more commonly nonputrid bronchiectasis is present. The pleural cavity is usually involved due to the contiguous extension from infection of the lungs, so that bronchopleural fistula, difficult to close, often accompanies the empyema. It is of interest that none of our patients has developed tuberculosis —an observation which may be related to the collapsed condition of the lung.* Rarely, large cystic dilatations filled with blood or straw-colored fluid occur in the lung. (Fig. 4A and B.) Pneumococcic infections occur in the obstructed lobe and give rise to a typical lobar pneumonia.

NATURAL AND THERAPEUTIC LIFE HISTORY

The natural growth of these tumors is slow. They take as long as twenty years to reach the size of a grapefruit. They invade by pressure atrophy and not by the true invasiveness exhibited by malignant, metastasizing tumors. By so doing, the bronchial wall is "penetrated." The extrabronchial portion grows to a larger size than the endobronchial portion. Extrathoracic metastases are unknown; mediastinal lymph-node metastases are exceedingly rare. Mediastinal encroachment may occur but phrenic paralysis, Horner's syndrome, or vena cava obstruction are not found. Growth of the tumors after various therapeutic procedures again indicates the very low growth potential of bronchial adenoma. After bronchoscopic removal, "recurrence" may be discovered as long as four to twelve years later. After biopsy through the bronchoscope, the wounds heal quickly. Transsection of the tumor during lobectomy has not produced untoward results. There is no evidence that the various surgical manipulations produced more rapid growth, metastasis or implantation.

Contrary opinions have been expressed as to the invasiveness and metastasizing capacity of adenomas. In our experience, whenever metastases have occurred in patients with similar clinical histories but doubtful histopathology of biopsy material, postmortem examination has shown the growth not to be adenoma but to belong to the lymphangio-endothelial sarcomas, leiomyosarcomas, slowly growing carcinomas or other as yet unclassified groups.[4]

* In a recent case of slowly growing adenocarcinoma (possibly adenoma) published by Gebauer, an acute tuberculous abscess occurred in the obstructed lower lobe.

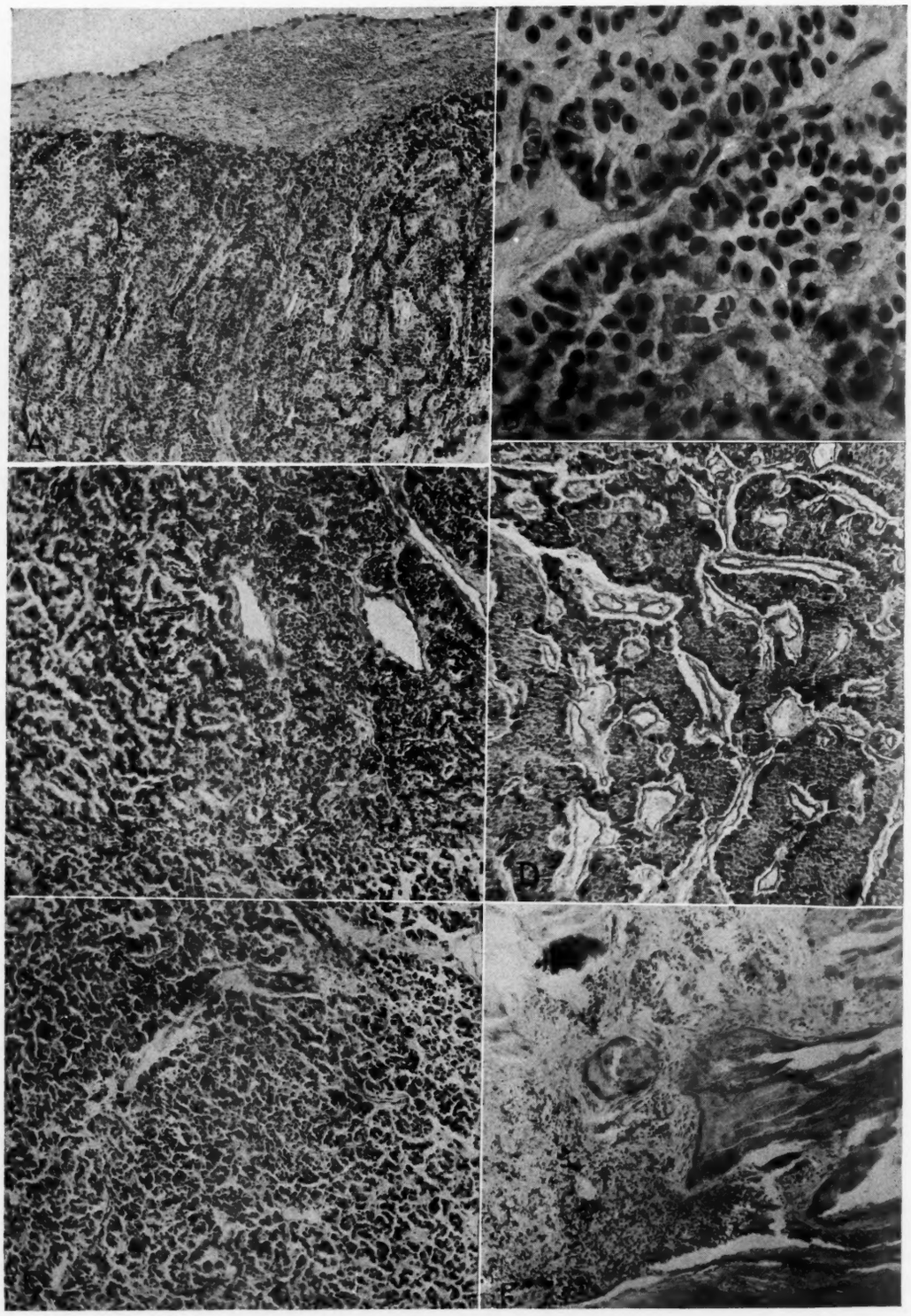


FIG. 3. Typical patterns obtained from postmortem or pulmonary resection specimens. A, Case XIV. G. D. Columns of uniform cells extending toward the fibrous tissue capsule. The bronchial mucosa has been denuded. Note the relative absence of pyknotic nuclei. × 120. B, Case XIV. G. D. Section of the surgical specimen showing both cylindromatous and alveolar patterns. × 750. C, Case IV. H. L. Section of the extrabronchial tumor. Note two different patterns on the same section. D, Case IV. H. L. Note the mosaic pattern made up of large groups of cells and prominent vascular stroma. This section of the tumor appears markedly different from that seen in C. × 120. E, Case I. I. K. Note the mosaic pattern made up of small groups of cells. This pattern was uniform throughout all the sections made of this tumor. × 120. F, Case X. F. J. Formation of osteoid tissue in extrabronchial portion of the tumor. × 120. (From Goldman, Alfred and Stephens, H. B. Polypoid bronchial tumors with special reference to bronchial adenoma. *J. Thoracic Surg.*, 10: 335, 1941.)

SYMPTOMS AND CLINICAL COURSE

Symptoms are related to location, vascularity and slow growth. Location of the adenoma in a major bronchus gives rise to symptoms of bronchial obstruction. The first symptoms result from disturbance in the transference of air in the large bronchial tubes. Wheezing, "asthma," irritating nonproductive cough, dyspnea, transient chest pains, "choked-up sensation" and respiratory postural discomfort may thus be the initial symptoms to appear. These are often so indefinite or transient as to be entirely overlooked unless they are inquired for very carefully. When the air becomes completely shut off from the alveoli distal to the tumor, atelectasis results; but when air is entrapped, emphysema occurs.

Bronchial drainage is also interfered with by the bronchial obstruction; consequently, the next symptoms to appear are those of pulmonary suppuration. Recurring pneumonias or so-called "drowned lung" are most common, but empyema, abscess, or bronchiectasis is frequent. The suppurative sequelae may be acute and dangerous, even giving rise to metastatic brain abscess. When death occurs, it usually results from the suppuration. The bacteriology of the suppuration is varied, but may include any one or several of the oral flora.

On the other hand, the slow growth of these tumors allows time for permanent chronic changes in the lung and pleura. These chronic inflammatory changes produce permanent structural defects which remain even after local removal of the tumor. In some cases, these structural changes in the lung are intimately related to the tumor, so that the tumor could not be removed without the removal of the outer surrounding lung. (Fig. 4A and B.) In others, the tumor lies in the stem bronchus and has no immediate relation to changes in the distal lung. As time goes on, the virulence of the infection within the obstructed lung dies down and the patients exhibit little if any toxicity and appear in good health. Such patients are frequently diagnosed pulmonary tuberculosis. In these people an astonishing degree of resistance seems to be acquired so that the smouldering infection, which may involve even an entire lung, flares up less often and produces little disability. (Fig. 5A and B.)

The cardinal symptom of bronchial adenoma is pulmonary hemorrhage, which is associated with the extreme vascularity of these tumors. They are characteristically sudden in onset and termination, bright red in color, profuse even to the extent of producing shock, unprovoked by cough or exercise, and in women frequently occur during the menstrual period. This type of hemorrhage probably arises from the tumor itself and is fed from the bronchial circulation. A second type of hemorrhage, composed of dark blood, often clotted and mixed with pus, induced by cough and exertion, and followed by blood-streaked sputum for several days, may occur. This type probably arises from the distal suppurating lung, and is fed from the pulmonary circulation. Older tumors may continue to cause hemorrhage even though the suppurative symptoms and toxemia have died down.

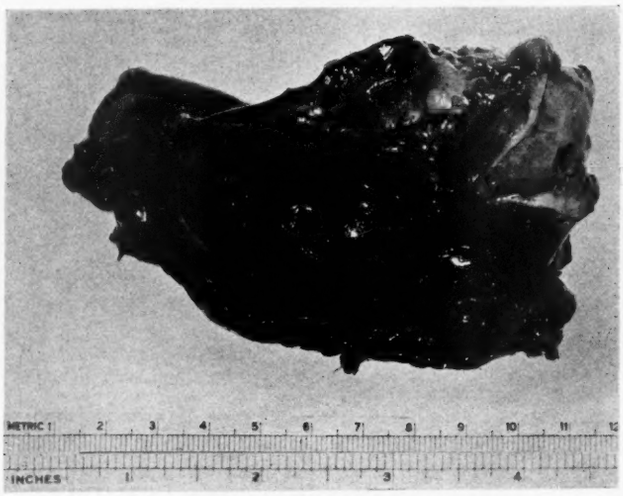
ROENTGENOLOGICAL FINDINGS

Roentgenologically, the appearance is bizarre (Fig. 5B) but characteristically lobar atelectasis is present and varying degrees of emphysema and atelectasis may occur. A marked shift of the mediastinum, thick pleurae, cystic whorls and abscess cavities overlie the tumor (Fig. 6A, B, C, D, E, F and G), so that it is rarely seen in the plain *x*-ray films. For the same reasons the extrabronchial portion and true extent of the tumor fail to be demonstrated. In an occasional case in which the tumor arises from a branch bronchus and is not surrounded by inflammatory density, a lobulated or rounded tumor mass may be visualized in an *x*-ray film. Such a mass may simulate a metastatic deposit rather than a primary tumor.

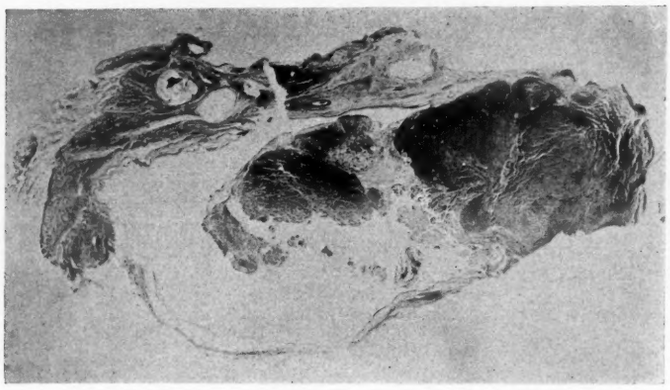
Tomography may delimit the true morphology, the relation to surrounding structure and the extent of the extrabronchial

portion when other roentgenologic studies fail. Tomographic findings, taken together with a bronchoscopic examination, are a great aid in differentiating adenomas from carcinomas. Tomography is a new but most important diagnostic aid and this procedure should be performed routinely, particularly if adenoma is suspected. Shadows cast by areas of occult atelectasis blend with the mediastinum so that they are easily overlooked in the plain *x*-ray film but are strikingly depicted in the tomogram.

Bronchography, particularly with endobronchial probing,[5,31] is valuable in determining the level of the bronchial obstruction and the condition of the distal bronchi. When lipiodol is prevented from flowing into the distal lung before endoscopic removal of an adenoma, a repeated bronchogram afterwards frequently discloses marked bronchiectasis in the distal lung. Initial bronchograms are characterized by a wide column of lipiodol with a smooth concavity at the distal end, and in the lateral view a filling defect is seen along one border.



A



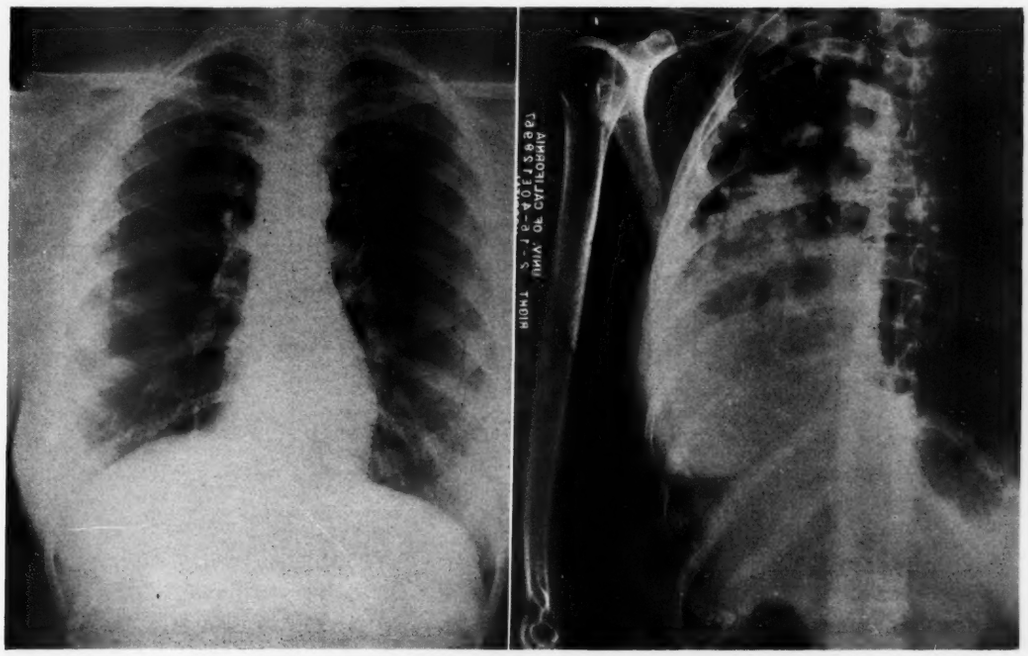
B

FIG. 4. Case XIX. E. A. A, lobectomy specimen of right middle lobe, showing tumor protruding through the cut right middle lobe bronchus. This patient had a duration of symptoms of approximately ten years. B, Microphotograph X 4 of section through the center of the entire specimen. Note that the tumor mass was projecting from a large cystic dilatation into the right middle lobe bronchus. This cystic dilatation was filled with hemorrhagic fluid. The surrounding lung is atelectatic, forms only a thin shell, and contains several bronchiectatic pockets. The histological picture was uniform throughout, and the pattern was similar to that seen in Figure 3E.

BRONCHOSCOPY

The bronchoscopist can readily determine the exact endobronchial location, the obstructed bronchi and the relation to the carina and trachea; but he can give only a partial answer to the nature of the bronchial attachment, the amount of bronchial distention or the suppuration in the distal lung, and he can give no answer as to the extent of the extrabronchial tumor. The failure of the bronchoscopist to realize his limitations has led to the erroneous conclusions that most adenomas were wholly endobronchial.

The bronchoscopic image is that of a soft or firm mass or polyp, whitish, pink or purple. These tumors are covered with mucosa and occasionally vessels traverse the bosselated surface. Their attachment to the bronchial wall varies from a few millimeters to 2 cm. in diameter; and when this tumor can be removed endobronchially, its stalk may be seen descending between the cartilaginous rings. Most often, however, the bronchus around the tumor is distended and its cartilages are thinned out or even absent. Frequently, the endobronchial portions of these tumors grow in a proximal direction toward the trachea so that they fill the entire length of a stem bronchus. When they arise near the bifurcation of a stem bronchus, lobulations may extend into the branch bronchus on either side.



A B

Fig. 5. Case II. J. B. A, March 19, 1928. Posterior anterior view, taken two weeks following bronchoscopic removal of adenoma. All the tumor was thought to have been removed. B, February 15, 1940, twelve years after bronchoscopic removal. Recurrence, with complete obstruction of the right stem bronchus has taken place. The right lung is now converted into a cystic lung. General condition good. Tomograms showed extra- as well as endobronchial tumor mass.

The bronchoscopic picture of an adenoma is usually distinctive enough to differentiate it from that of a primary bronchial carcinoma. In the latter there is ulceration and induration with the characteristic "frozen" condition of the surrounding structures, and the carina may be narrowed in the anterior posterior diameter from metastatic lymph glands in the hilar area. Adenomas, on the other hand, may be freely movable. Ulceration usually is not present and there is no infiltration of the adjacent bronchial mucosa. Occasionally, when the adenoma presents as low, broad

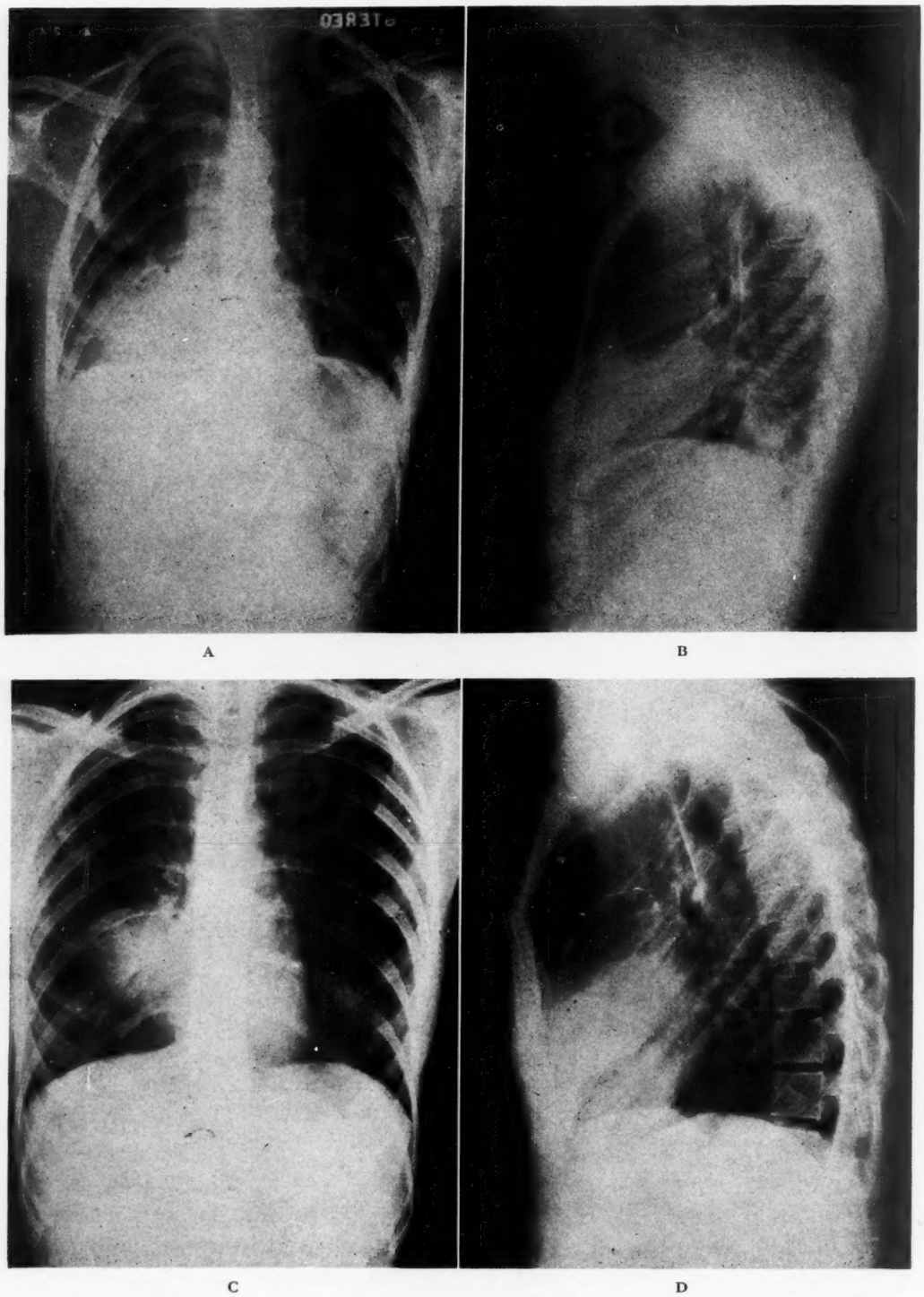
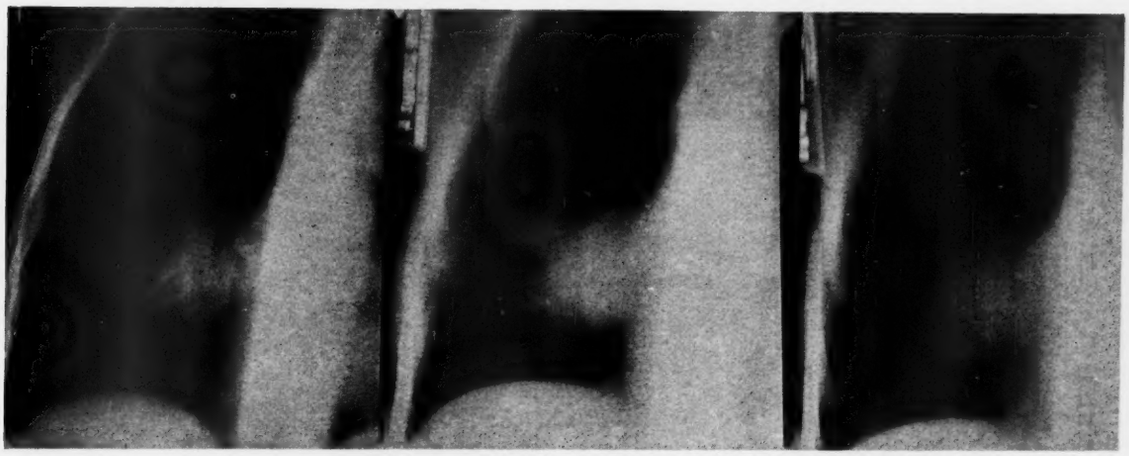


FIG. 6. Case XIX. E. A. A and B, roentgenograms showing dense shadows at the right base occupying the right middle lobe. When compared with the surgical specimen, the actual size of the tumor is much smaller than the shadow cast. C and D, nine months later. The shadow seems slightly smaller; very little change.

induration instead of a polypoid mass, the bronchoscopic differentiation is impossible. Hemorrhage gives another important bronchoscopic clue to the true diagnosis. We have frequently encountered rather severe bleeding from the adenomas with the least trauma, whereas the same amount of bleeding is unusual in the case of polypoid carcinoma.



E F G

Fig. 6. E, F and G, tomograms 8, 10 and 12 cm. from the back. Note that because of the fluid in the cyst which surrounds the adenoma even tomography fails to delimit the true size of the adenoma.

Bronchoscopic removal of adenoma reestablishes the bronchial airway and also the drainage of distal infection, thus bringing about a dramatic improvement of the patient. But due to the intramural and extrabronchial extensions of these tumors, it is doubtful that every cell is removed by such treatment. Consequently, late recurrences can be expected frequently, although they may not be suspected for ten years or more. (Case 11, J. B.) For this reason, patients treated bronchoscopically should always be followed by bronchoscopic examinations at intervals of no less than one year. In addition, the older tumors are so large and adherent to the thinned-out bronchial walls that endobronchial removal is impossible and may bring about an unnecessary fatality.

DIFFERENTIAL DIAGNOSIS

The finding of tubercle bacilli rules out adenoma because we have yet to see a patient with bronchial adenoma develop a positive sputum. Bronchoscopy, of course, usually tells the story. Bronchial tuberculosis presents a flat ulcerative process, smears from which show acid-fast bacilli; while adenoma projects as a tumor mass, biopsy of which may be characteristic. Because carcinoma and other malignant tumors may present as a polypoid tumor to the bronchoscopist, adequate biopsies with careful histologic interpretations are frequently necessary to bring about early diagnosis. Gebauer[32] has called attention to the endobronchial morphology of certain adenocarcinomas which metastasize widely. We have encountered difficulty in differentiating peripheral circumscribed epidermoid, cylindrical ciliated adeno-, undifferentiated and unclassified carcinomas. Certain connective tissue tumors, such as angio-endotheliosarcoma and myosarcoma, are apt to be of long duration and polypoid in nature, and for many months they may simulate adenoma. A careful and complete study of the patient, particularly with tomographic studies, may disclose the invasive nature of these tumors. Adequate biopsies containing characteristic patterns make the diagnosis certain. Bronchoscopic biopsy material from small-cell carcinoma and angio-endothelioma has proved difficult to differentiate from adenoma. Exploratory thoracotomy should not be delayed too long when the diagnosis of

adenoma cannot be substantiated in other ways. Benign bronchial tumors other than adenoma are much less common than was previously thought. Fibromas occur only about one-tenth as frequently as adenomas, and the characteristic patterns of adenomas are usually lacking in these. Ecchondromas, myomas and lipomas are also rare and have a characteristic histology. Occasionally, unclassified benign polypoid tumors occur.

Bronchiectasis is not always of the fetid type with marked cough and sputum. The hemorrhagic type of bronchiectasis may simulate the clinical picture of adenoma. When bronchiectasis has been demonstrated and hemorrhage is the chief symptom, the presence of a concomitant adenoma should be kept in mind. In such cases, bronchoscopic visualization and biopsy of the adenoma may be difficult. Tomographic studies may be of help.

TREATMENT

There are three types of treatment for bronchial adenomas: local treatment, radiation and pulmonary resection.

The local treatment may be accomplished endoscopically or by local resection through a transpleural approach. The endoscopic treatment is less commonly indicated than it was formerly thought to be, because of (1) local recurrences, (2) danger of fatal complications, (3) inability to remove adequate amounts of the tumor, and (4) disabling symptoms from the distal suppurating lung still remaining. It must be admitted, however, that where adequate bronchial drainage can be re-established by removal of the endobronchial tumor, improvement is often satisfactory; and, even though late recurrences result, the general condition of these patients may remain good for as long as twelve years. Yet even these patients eventually change from a problem of bronchoscopic therapy to one of pulmonary resection. In preparation for pulmonary resection, re-establishment of bronchial drainage by endoscopic removal of the obstructing tumor should be performed whenever it is easily accomplished. But when these attempts are complicated by serious hemorrhage, failure to establish bronchial drainage easily or the presence of persistent obstructing tumor, then bronchoscopic treatment should be abandoned in favor of pulmonary resection.

The number of patients operated upon by transpleural approach with local resection of the tumor through an incision in the bronchus is still quite small. This method appears to be applicable to those tumors in which the distal lung has not yet been damaged permanently, in which the size is small and in which dissection is easy.

Radiation therapy probably has little effect upon the tumor *per se*. When distal infection is pronounced and bronchial obstruction complete, external radiation may produce an exacerbation of the symptoms. It is our belief that those patients who had radiation therapy continued to live, not because of the therapy, but rather in spite of it. In our hands, intrabronchial radiation therapy has proved too cumbersome and dangerous. Besides, the remaining untreated suppurating lung is apt to be the chief cause of disability and symptoms so that a cure is not obtained.

Pulmonary resection, then, is the treatment of choice in approximately 90 per cent of the patients. It accomplishes not only the complete removal of the tumor but also of the suppurating lung. Unlike carcinoma, lobectomy rather than pneumonectomy is sufficient if it includes all the tumor and the suppurating lung. Mediastinal lymph-node dissection is rarely necessary. Preoperative preparation consisting of transfusion, rest, high caloric, high vitamin diet, and release of bronchoscopic obstruction by endoscopic therapy, is important to keep the operative mortality low. Operation should not be performed during an exacerbation of the pulmonary infection or immediately after a large hemoptysis. Several weeks or months may be utilized to raise the patient's preoperative condition to the optimum.

In general, the technic of lobectomy for bronchiectasis (Brunn,[33] Churchill[34]) with hilar ligation of vessels and primary liga-

tion and suture of the bronchial stump, as described by Blades,[35] is the method of choice. Certain peculiarities of these tumors must be considered, namely, the enlargement of the bronchus due to the tumor within it, the location close to the stem bronchi, the tendency for the tumors to project towards the trachea and the adhesive pleuritis associated with a long infection. The enlargement of the bronchus disturbs the relation of the vessels so that they must be dissected out with great care at the hilum. The position of the tumors makes it necessary to amputate the lobe close to the stem bronchus. However, at times the incision in the bronchus may be made below the tip of the endobronchial tumor so that a slightly longer bronchial stump is obtained. The adhesive pleuritis frequently involves the pericardium and the adhesions may contain fairly large blood vessels. These adhesions must be ligated before they are cut.

In our series, seven of nineteen patients have had pulmonary resection performed. Three of these underwent pneumonectomies and four lobectomies. There was one operative mortality in a patient who had a pneumonectomy. In the remaining six cases the results are very satisfactory. Wherever the tumor can be removed with lobectomy alone, this should be done.

CONCLUSIONS

Bronchial adenomas are epithelial polypoid tumors which exhibit growth potentialities midway between distantly metastasizing carcinomatous and purely local polypoid bronchial tumors.

Three morphologic types occur: endobronchial, intramural and extrabronchial. The incidence of intramural and extrabronchial tumor is at least 90 per cent.

Bronchial adenomas have a characteristically long clinical course which is related to the location in the large bronchus, their vascularity and their slow growth.

Histologic examination of bronchoscopic biopsy will definitely establish the diagnosis only if characteristic patterns are present.

Tomography offers a diagnostic procedure that can determine the location, extent and morphology of these tumors.

Endoscopic removal of bronchial adenomas is indicated to reestablish bronchial drainage; only in the rare endobronchial type with thin pedicle can this type of removal be complete.

Pulmonary resection may be indicated because of growth of the endobronchial portion and the suppuration in the distal or surrounding lung in approximately 90 per cent of the patients.

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BRONCHIAL OBSTRUCTION*

SIGNS, SYMPTOMS AND DIAGNOSIS

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BRONCHIAL obstruction is a fundamental pathological process which plays an extensive, variable rôle in pulmonary pathology. Its influence on the lung is usually determined more specifically by the location of the lesion and the degree to which the bronchus is obstructed, than by the etiology of the process which has produced the obstruction. The effects of an obstruction of a bronchus may be widespread, and not limited to the lung distal to the obstruction. Gross, significant changes in intrathoracic pressure, circulatory changes, extensive suppurative processes and other physiologic and pathologic processes result. It is the purpose of this paper to discuss principally the mechanics of bronchial obstruction and the signs and symptoms which it produces. The disease entities which may be responsible for the obstructions, as well as those which are produced by them, will serve as illustrations.

ANATOMY

A thorough understanding of the anatomy of the tracheobronchial tree and the topographic anatomy of the lobes of the lungs and their projected physical and roentgen findings is essential to establish the exact location of the point at which a bronchus is obstructed. Most anatomy texts do not provide the description or the terminology used by the bronchoscopist or the thoracic surgeon to correlate the site of an obstructing lesion in a bronchus with the peripheral distribution of the effect of the obstruction. The recent descriptions of Neil,[1] Hasse,[2] Churchill and Belsey,[3] de Pablo,[4] Jackson and Huber,[5] Adams and Davenport,[6] and others have done much to focus attention on the importance of the segmental arrangement of the lungs and the bronchi leading to them. A review of this anatomy is somewhat outside the scope of this paper and may be obtained directly from the published works of the authors mentioned.

PHYSIOLOGY

The functions of the tracheobronchial tree are three-fold: the conduction of air to and from the lungs, the final preparation of the air for the lungs, and the drainage of secretions and foreign material from the lungs. The manner in which the first of these functions is executed determines in a large measure the mechanics of bronchial obstruction. The tracheobronchial tree cannot be considered merely as a fairly rigid tubular system, but rather as a dynamic conductor system whose rôle is anything but passive. The bronchi dilate and constrict, and lengthen and shorten during inspiration and expiration. Further, they deviate inferiorly and superiorly as the lungs follow the excursion of the diaphragm with a total resultant motion simulating peristaltic waves.

The second function of the tracheobronchial tree, that of the final preparation of air for the lungs, is of relatively little direct significance to bronchial obstruction. It consists simply of warming and humidifying the air, and of removing foreign material held in suspension in the air.

The third function, that of drainage, is influenced more than mechanically by a

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bronchial obstruction. Normally, this function is carried out by a number of contributing factors, including the action of the cilia, the action of the glands of the bronchial mucosa liquefying secretions, the respiratory movements, the tussive squeeze of the cough reflex and finally the blast of the cough itself as the built-up intrabronchial and intratracheal pressure is released by a sudden opening of the glottis. Almost any degree of obstruction anywhere in the tracheobronchial tree will influence one or more of these actions, producing profound changes in bronchopulmonary physiology. These changes may be in the nature of a loss of control of intrathoracic air pressures, a further loss of the important relationship of these pressures to blood flow within the chest, and massive retentions of the products of glandular secretions with superimposed infections.

The discussion of the physiologic effects of respiratory obstruction should be amplified because their critical analysis may not infrequently result in the prevention of irreparable damage. Dyspnea is the first of these effects. It may be defined simply as an abnormal increase in breathing effort and its onset may cause one of three phenomena to take place: either an increased rate of ventilation or minute volume of air breathed, maintenance of the previous normal level of ventilation through a neutralization of the effects of the added ventilation by the increased effort expended, or a decrease in the ventilation to a level below normal. However, it must be remembered that the normal individual sitting quietly in a chair can double the rate of pulmonary ventilation with no obvious effort and that the changes would be recognized only by the trained observer. Therefore, in the dyspneic patient it is essential to evaluate the air movement in and out of the chest to determine whether a bronchial or tracheal obstruction is compensated by the dyspnea, or is decompensated and requires urgent relief in the form of oxygen or surgical intervention before the decompensation becomes irreversible.

The cause of dyspnea may be reflex or physiochemical, or a combination of these two factors. Reflex dyspnea is the result of abnormal kinetic afferent impulses arising in the muscles of respiration and in the musculature of the tracheobronchial tree through the Hering-Breuer reflex. These impulses are initiated by an impedence of the air flow or by a reduction in its velocity, and are responsible, together with certain physiological phenomena, for the subjective sensations which cause dyspnea. The physiochemical factor responsible for dyspnea in tracheobronchial obstruction is produced both by an increase in the carbon dioxide content of the blood, which stimulates the respiratory center, and by a decrease in the oxygen content of the blood, which increases the irritability of the respiratory center.

Great variability exists between individuals in their response to abnormal respiratory conditions. An occasional patient is seen whose dyspnea may be entirely of the reflex type and may become so severe that intervention is necessary even though respiratory decompensation has not occurred. In a similar way there may be a variation in response to the rise of carbon dioxide in the blood. Anoxia may develop gradually and be tolerated without great difficulty. However, with the sudden relief of a chronic tolerated anoxia, as may occur following a tracheotomy or the removal of an obstructing foreign body, the carbon dioxide may be exhaled so rapidly and the anoxia relieved so quickly that the lack of stimulation of the center and its decreased irritability may result in a respiratory paralysis and death. A similar action, less severe in degree, may follow the administration of oxygen in other types of respiratory decompensation. In chronic hypertrophic pulmonary emphysema, for instance, oxygen may cause severe psychotic symptoms which gradually improve as the therapy is continued.

In the diagnosis as well as in the management of obstructive diseases it is imperative to recognize the onset of respiratory decompensation as it manifests itself by the presence or the onset of anoxia. When anoxia starts, the metabolic processes in the body are impaired and muscular strength and ability are decreased. Intervention becomes imperative because the course is unfavorable and the point may soon be reached when relief may not be effective. The recognition of anoxia in the early stages is difficult and the only reliable although impractical method is by oxygen concentration determinations of arterial blood. Probably the most significant finding is the change in the minute volume of ventilation. When this is reduced and especially when it is combined with a change to rapid and shallower breathing, anoxia has started and extreme measures in the relief of the obstruction are indicated. Cyanosis is not always a reliable sign, either by its absence or its presence. A sudden increase or even decrease in the pulse or respiratory rate and a rise in body temperature out of proportion to a superimposed infection are significant signs. The most important single observation in the general appearance of the patient is the presence or absence of exhaustion.

Alterations in the intrabronchial and intrathoracic pressures are responsible for the second group of changes in physiology which follow respiratory obstructions. These alterations directly affect the blood flow through the chest, and the secretion of fluids into the alveoli and bronchi. Kernan and Barach,[7,8] Galloway,[9] Gailbraith,[10] Adams and Livingston,[11] and others have emphasized these effects and have reviewed the related literature. The entire subject of experimental bronchial obstruction is so extensive that in this discussion the findings can only be summarized. It has been shown that the increased positive pressure of expiration has no appreciable effect on the blood flow. However, the increased negative pressure on inspiration has definite harmful effects which may be summarized as follows: (1) Negative intrathoracic pressure results in an increase in the pulmonary capillary blood pressure

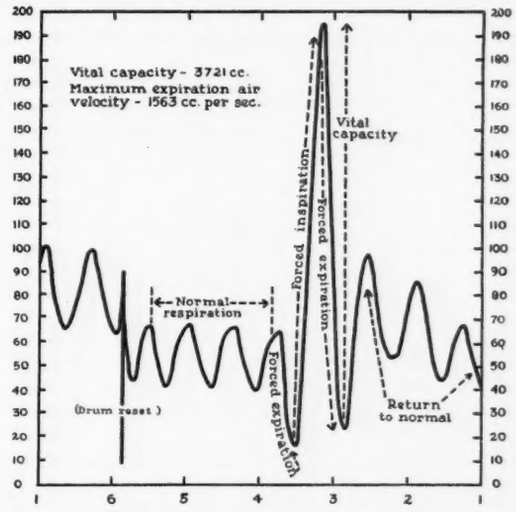

Fig. 1. Normal respiratory curves recorded on a high speed recording drum.

with transudation of serum into the alveolar spaces. This is a direct result of the increased negative pressure on the heart and circulation. The high negative intrathoracic pressure increases the return flow of blood to the heart so that the blood flow through the chest is increased. The flow of blood from the intrathoracic to the extrathoracic aorta and large arteries is likewise hindered by the negative pressure, thus putting an additional load on the left ventricle. As the negative pressure rises these two effects just mentioned increase until a progressing accumulation of blood occurs in the lungs causing the rise in capillary blood pressure with congestion, transudation, and pulmonary edema. (2) Exudation of fluid into the alveolar spaces and bronchioles because of the suction action of the intrabronchial and intra-alveolar negative pressure. (3) A vicious cycle producing a further increase in the negative pressure due to the attempt to compensate for the effects of the other two actions by increasing the respiratory effort.

Thus, the physiologic effects of tracheobronchial obstruction may be divided into their respiratory and their cardiovascular actions. Mild obstruction results in a dyspnea which remains compensated by

reflex and physiochemical stimulation. Severe obstruction results in respiratory decompensation, or anoxia. The cardiovascular phenomena manifest themselves by a pulmonary edema and finally a circulatory failure due to rising intrathoracic negative pressures.

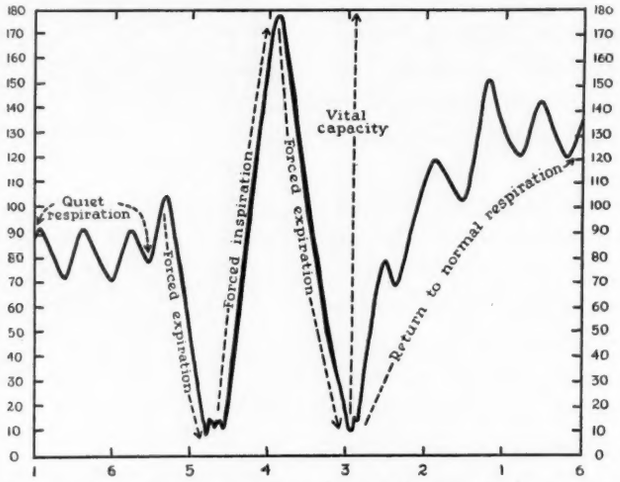

FIG. 2. Respiratory curves of a patient with laryngeal obstruction. The uniformity of the velocity of forced expiration and forced inspiration and their slow rates are the characteristics of this type of obstruction.

The actual measurement and mathematical interpretation of the degree or character of a bronchial obstruction is based upon the physiology of the respiratory exchange. The supply of oxygen to the alveoli and the removal of carbon dioxide is accomplished by two different mechanisms: the tidal air, i.e., the air which moves in and out of the chest with each respiration, serves as a ventilating mechanism. The air stream does not actually enter the alveoli except in extremely high ventilation. The second and most important mechanism of respiratory exchange is through diffusion. These two mechanisms allow the alveolar air to be fairly constant in its oxygen and carbon dioxide concentrations throughout inspiration and expiration so that the exchange of the gases between the blood and alveolar air goes on continuously and quite uniformly.

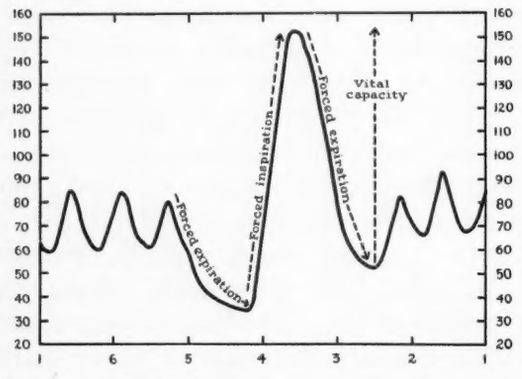

FIG. 3. Respiratory curves of a patient with bronchial asthma. Note the reduced velocity on expiration, the long curve of forced expiration contrasting sharply to the straight curve of rapid, regular inspiration.

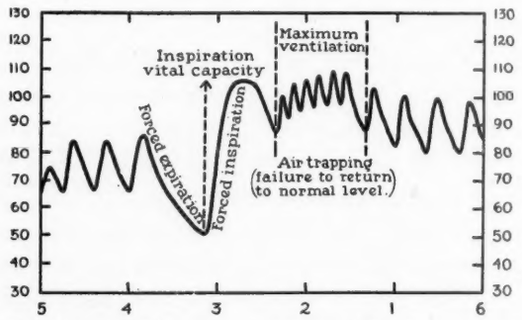

FIG. 4. Tracings of a patient with obstructive pulmonary emphysema. The characteristics of this curve are a slow, rather uniform velocity of expiration, and a trapping of air following forced inspiration.

The functional examination of respiration is a clinical method of spirometry which records graphically the movement of air in and out of the chest. The Benedict-

Roth metabolism apparatus modified by the addition of a high speed drum is the apparatus of choice and the technic has been described by Barach[12] and Cournand[13] and their co-workers and by Andrews.[14] This method records the ventilation and tidal air, and also reveals the qualitative characteristics of respiration which are of diagnostic significance in respiratory obstructive diseases. (Fig. 1.)

Respiratory obstruction in the larynx and trachea shows a symmetrical slowing of inspiration and expiration at the extremes of the vital capacity tests. (See Fig. 2.) Bronchial asthma (Fig. 3) is characterized by a curved expiratory tracing and obstructive emphysema (Fig. 4) by a moderately slowed expiratory velocity and trapping of air on successive maximum expiratory efforts. Such examinations can be used not only in the differential diagnosis of these various types of obstructive diseases, but also to demonstrate the effectiveness of therapeutic procedures such as oxygen, helium, positive pressures, and the changes after the administration of drugs.

ETIOLOGY

A division of the lesions responsible for bronchial obstructions into intrabronchial, endobronchial and extrabronchial groups serves as an etiologic classification. Indirectly, the age of the patient must likewise be considered a factor influencing the pathological condition produced by a bronchial obstruction. The small caliber of the trachea and bronchi of infants is responsible for extensive pulmonary changes produced by lesions which would be insignificant in the air passages of a child or an adult. Similarly, the very soft nature of the cartilage of the trachea and bronchi of infants permits easy compression of the airway which would be impossible in the more rigid bronchi of adults. The fundamental mechanics of the changes produced by bronchial obstruction in infants, however, are identical to those found in adults.

Intrabronchial obstructions present the most striking and illustrative examples of the various effects of bronchial obstruction. Into this division fall the cases of intra-

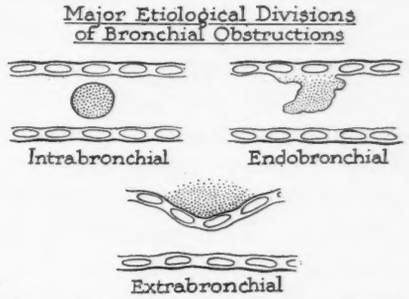

Fig. 5. Diagrammatic representation of bronchial obstruction produced by intrabronchial, endobronchial and extrabronchial lesions.

bronchial foreign bodies,[15] postoperative massive collapse of the lungs,[16] the bronchial obstructions produced by exudates in laryngotracheobronchitis,[17] and atelectasis produced by the thick plugs of asthma.[18] Intrabronchial obstructions may be further subdivided into endogenous and exogenous types. Broncholiths, thick, tenacious, bronchial secretions, inflammatory exudates, and occasionally the material from a lymph-node which has eroded through the bronchial wall, comprise the endogenous obstructions. Foreign bodies of every description make up the large list of exogenous elements which produce intrabronchial obstruction. The recognition and description of many phases of the whole problem of bronchial obstruction, with studies of its physiology and morbid anatomy were, in a large measure, possible through clinical and experimental studies of the effects of these foreign bodies in the air and food passages.

Endobronchial obstructions are due to lesions of the bronchial mucosa, musculature or cartilagenous wall. They have assumed a constantly increasing importance as bronchoscopy and bronchography have demonstrated not only their presence but also their effect on bronchopulmonary physiology. They consist of inflammatory swellings or cicatricial stenoses of the mucosa which partially or completely occlude the bronchial lumen; the various

types of infiltrative, ulcerative, granulomatous or cicatricial tuberculous lesions;[19,20] the specific inflammatory lesions of syphilis, rhinoscleroma and leprosy with the characteristic deforming scars which these lesions produce when healing;[21] and both benign and malignant neoplasms. The distortions produced by the kinking or twisting of the bronchial walls following such procedures as prolonged artificial pneumothorax, thoracoplasty or injuries must likewise be included in this group, although they should not be confused with compressions of the walls for which many of these same factors may be responsible.

The extrabronchial factors of bronchial obstruction consist of lesions in the mediastinum, heart or lungs which compress the lumen of a bronchus by pressure from outside the bronchial walls. Of these, enlarged tracheal and hilar lymph-nodes, both tuberculous and nontuberculous, are the commonest of the non-neoplastic lesions. These nodes at times assume an extreme size and compress the trachea or both bronchi to the point of asphyxia. Enlarged mediastinal nodes are fairly common in children[22] in whom, because of the soft nature of the cartilagenous bronchial wall and the relatively small size of the lumens of the major bronchi, severe dyspnea may be produced by a relatively mild degree of enlargement of the nodes. Other extrabronchial lesions producing bronchial compression consist of pulmonary and mediastinal tumors, aneurysms, cardiovascular anomalies, esophageal tumors or even occasionally esophageal foreign bodies.

The following is an outline of the etiology of bronchial obstruction: (See Fig. 5.)

I. Intrabronchial Obstructions
- *A.* Endogenous
  1. Secretions
  2. Exudates
  3. Broncholiths
  4. Contents of ruptured lymph-nodes
- *B.* Exogenous
  1. Foreign bodies
  2. Aspirated stomach contents

II. Endobronchial Obstructions
- *A.* Congenital web
- *B.* Nonspecific inflammatory processes
  1. Edema
  2. Infiltration
  3. Cicatricial stenosis
- *C.* Specific inflammatory processes
  1. Tuberculosis
     a. Edema
     b. Infiltration
     c. Granulomas
     d. Ulceration
     e. Stenosis
  2. Syphilis
  3. Rhinoscleroma
  4. Leprosy
- *D.* Distortion of the walls
  1. Kinking
  2. Twisting
- *E.* Neoplasms
  1. Benign
  2. Malignant

III. Extrabronchial Obstructions
- *A.* Inflammatory
  1. Enlarged lymph-nodes
  2. Mediastinal abscess or phlegmon
  3. Lesions of the vertebra
- *B.* Cysts
  1. Pulmonary
  2. Mediastinal
- *C.* Emphysema
- *D.* Neoplasms
  1. Mediastinal
  2. Pulmonary
  3. Esophageal
- *E.* Cardiovascular
  1. Aneurysm
  2. Heart
     a. Dilatation of left auricle
     b. Enlargement (congenital heart disease)
     c. Anomalies
- *F.* Esophageal foreign bodies

As with any type of classification, it is difficult to separate or segregate any of these factors in an absolute manner. A number of overlapping manifestations occur with many of the lesions, and it may be

impossible to determine the one most responsible for an obstruction in any given case. For instance, an endobronchial tumor may partially obstruct a bronchus, but total obstruction of the bronchus may follow because of compression by the extrabronchial portion of the tumor.

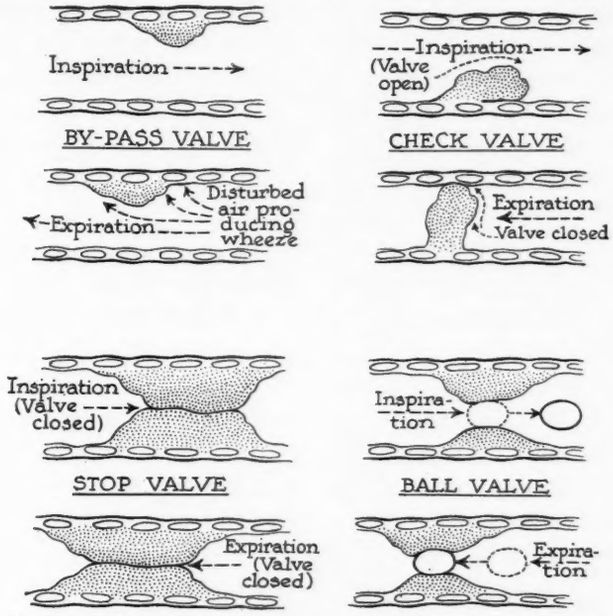

Fig. 6. Diagrammatic representation of the manner in which the four types of valves act to produce bronchial obstruction.

TYPES OF BRONCHIAL OBSTRUCTION

Important as are the factors which produce bronchial obstruction, it is the degree of the obstruction and its location which are responsible for the pathological condition of the lung beyond the point of obstruction. In the majority of instances, the mechanics of obstruction can best be described by designating the obstructions as valves in a system of tubes. (See Fig. 6.) As the Jacksons[23] point out, however, these tubes are only semi-rigid and they are not of constant caliber. Inspiration is accompanied by an expansion of the bronchus and expiration by a contraction. Consequently, numerous factors enter into the mechanics of each particular obstruction, but three types of phenomena form the basic physical effects of bronchial obstruction. These are well known but will be briefly reviewed. Their mechanics can best be illustrated by a model such as that which Eloesser and Freeman[24] have constructed. The simplest, or by-pass valve, consists of a small decrease in the size of the bronchial lumen such as might be caused by an object lying across the diameter of the bronchus, the lumen remaining large enough to allow air to pass easily to and fro, without appreciably changing the volume of air entering or leaving the lung beyond the point of obstruction. Air passing back and forth over this irregularity is not actually obstructed except in that the column of air is broken to form eddies. Such an obstruction may be produced by a small mucous plaque on the bronchial wall, a small irregularity of the bronchial wall due to an early bronchogenic carcinoma, or a foreign body, such as an open safety pin lying loosely in the bronchus. The only evidence that can be obtained of the presence of such an obstruction is a limitation of motion of the chest on the affected side[25] and a wheeze heard at the open mouth or on auscultation over the chest, loudest at the point of obstruction.

The second type of bronchial obstruction is observed when the obstruction has become more marked. It is characteristically

produced by many foreign bodies such as nut kernels, and is present at some stage of the growth of most endobronchial tumors or cicatricial bronchial stenoses. It is designated as a check-valve or ball-valve type of obstruction because air may pass in one direction only, and is dependent upon the physiological bronchial expansion and contraction which occur during normal respiration. Thus, if a foreign body lies in the bronchial lumen, air may pass it freely during inspiration as the bronchus increases in size. During expiration, however, the bronchial wall collapses around the foreign body, obstructing the flow of air out of the lung distal to the foreign body. Another factor contributing to this type of expiratory obstruction is the fact that the force of inspiration is greater than that of expiration. This difference increases with respiratory embarrassment. On subsequent respirations, as more and more air becomes trapped, an emphysema develops in the lung distal to the obstruction. This may be observed by physical signs, on the fluoroscopic screen or on roentgen films taken at inspiration and expiration. The classical signs are a shift of the heart and mediastinum toward the uninvolved side on expiration with a return to the normal position on inspiration and a stationary, depressed diaphragm, emphysema, and widened and immobile intercostal spaces on the involved side.

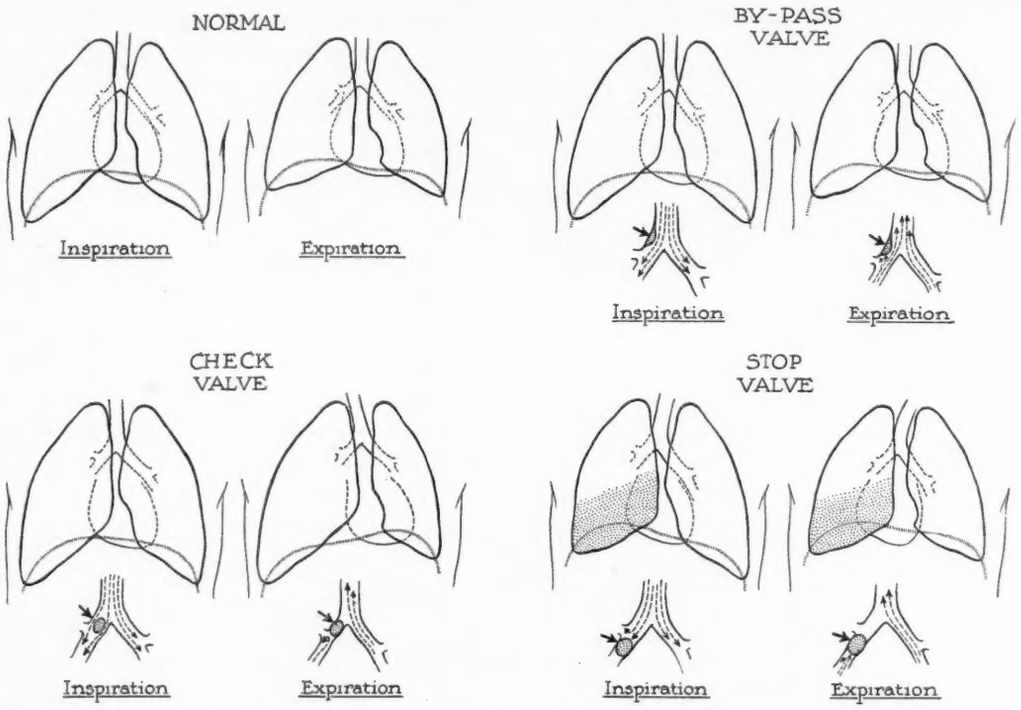

Fig. 7. Positions of the lungs, heart, trachea and diaphragm during inspiration and expiration in the various types of bronchial obstruction.

It is possible, of course, that this type of obstruction may involve only the bronchus leading to a single lobe, and thus may produce a marked emphysema of one lobe alone or a cavity simulating a lung abscess.[26,27] Similarly, if the bronchiole leading to a lobule is thus involved, a bullous emphysema may occur, which may rupture and produce a pneumothorax.[28] An emphysema extending under the visceral pleura and into the mediastinum may also follow such an obstruction.

A difference between the mechanics of the check-valve and the ball-valve does exist. However, their ultimate action in regard to bronchial obstruction is, in most instances, identical. In the check-valve, the valve seat remains in a constant position, opening and closing as the bronchus expands and contracts. In the ball-valve,

the obstructing element moves back and forth during inspiration and expiration, moving into and out of the valve seat. Under certain conditions, the check-valve or the similar ball-valve acts in the reverse manner, allowing air to leave but not enter a bronchus. Under such conditions, the lung rapidly becomes airless or atelectatic and the signs and symptoms are those of atelectasis.

Complete obstruction of a bronchus, allowing no air to pass the obstruction either on inspiration or expiration, is designated as a stop-valve obstruction. Following such an obstruction the air in the portion of the lung beyond is absorbed and the lung becomes airless and shrunken, or atelectatic. A typical example of such an obstruction is the complete occlusion of a bronchus by a round foreign body such as a nail head, or by an extensive, fungating bronchogenic carcinoma. The typical findings which characterize such an atelectasis are the density of the area beyond the obstruction, the shift of the heart and mediastinum toward the involved side, the elevation of the involved diaphragm and narrowing of the intercostal spaces on the involved side. The opposite lung, if the atelectasis is extensive, shows some degree of compensatory emphysema.

Figure 7 illustrates the position of the heart, diaphragm, trachea and lungs during inspiration and expiration in each of these types of bronchial obstruction.

SIGNS AND SYMPTOMS

From the foregoing discussion, it is apparent that three principal factors determine the signs, symptoms and ultimate pathological condition of an obstruction of the lower air passages. The first of these is the level in the tracheobronchial tree at which the obstruction occurs; whether it is in the trachea, a major bronchus, or a bronchiole. Grossly, this may be considered as an *anatomical* factor. The second principal factor is the degree to which the air passage is obstructed; i.e., whether the action produced is that of a by-pass valve, check-valve, or stop-valve. This factor is a *functional* one. Finally, the signs and symptoms will be dependent, but to a lesser degree, upon the relation of the obstruction to the bronchial walls; whether it is an intrabronchial, endobronchial, or extrabronchial obstruction. This is an *etiologic* factor. As these factors act collectively to produce bronchial obstruction they must be considered as they collectively produce the various signs and symptoms of bronchial obstruction.

The symptomatology of bronchial obstruction is for the most part not characteristic of bronchial obstruction *per se;* certain underlying symptoms are fairly constant, however, influenced in each case by the underlying etiologic factor. Other symptoms become important only when they are associated with a history of a bronchopulmonary pathological condition. A cough, suggestive of bronchial irritation, may not in itself be significant, but it is one of the most constant symptoms of bronchial obstruction. It may be dry and entirely unproductive for weeks and suddenly be associated with hemoptysis and a foul, purulent sputum produced by a superimposed infection.

A wheeze is also an extremely important symptom because, as stated above, an object lodged in a bronchus permitting air both to enter and leave the bronchus freely will produce none of the changes in the lung distal to the obstruction which are so characteristic of the other types of bronchial obstruction. Thus, with no abnormal physical findings, the wheeze heard throughout the chest, loudest over the location of the obstruction, may be the only sign or symptom of the object if it is nonopaque to *x*-rays.

Dyspnea is likewise an outstanding symptom of tracheal or bronchial obstruction. All degrees of dyspnea may be noted from the rapidly fatal dyspnea accompanying an obstruction at the bifurcation of the trachea to the sensation of constriction of the chest, or chest discomfort which is a characteristic expression used by patients

with early bronchial neoplasms. Pain is not a common symptom unless the underlying pathological condition is extensive and associated with destructive changes. Patients with sharp foreign bodies in the bronchi rarely complain of pain, although irregular objects such as sharp bone fragments or dental fillings occasionally cause pain which the patient may localize quite accurately to the site of the foreign body.

Certain symptoms should be mentioned as characteristically accompanying lesions that produce bronchial obstructions. The enlargement of the neck and venus engorgement of the neck and chest which follow upper mediastinal neoplasms are examples of such symptoms. Similarly, the hoarseness due to a paralysis of the left vocal cord following involvement of the left recurrent laryngeal nerve in a neoplastic process at the hilus is a symptom of that type of neoplasm.

Hemoptysis is one of the most important symptoms of any type of pulmonary pathological condition. In reference to bronchial obstruction it is not infrequently the first evidence of the presence of an intrabronchial foreign body or an early bronchogenic carcinoma. Benign bronchial neoplasms have been known to manifest themselves by this symptom many months before their slow growth occludes the lumen of the bronchus to give further evidence of their presence.

PHYSICAL FINDINGS

In spite of the extremely important rôle which roentgenology plays in the diagnosis of diseases of the chest, the physical examinations in themselves not infrequently lead to important clues which finally establish the correct diagnosis, in spite of contrasting or negative roentgen findings. One of the most constant signs in almost all cases of bronchial obstruction is a definite limitation of motion on the involved side, independent of the degree of the obstruction. Careful inspection of the chest is thus of great diagnostic importance in spite of its apparent simplicity. Other signs and symptoms are more dependent upon the degree of the obstruction and on its location.

TRACHEAL OBSTRUCTION

Tracheal obstruction may manifest itself by all the signs and symptoms of acute respiratory obstruction, leading to a rapid fatality; in the other extreme, it may manifest itself only through the simplest sign of obstruction to the airway, a wheeze. Foreign bodies loose in the trachea may be heard flying back and forth from the larynx to the carina during respiration, a finding which Jackson[23] has designated as an "audible slap" which is accompanied by a "palpatory thud" if one palpates the larynx. The "asthmatoid wheeze" is invariably present in such obstructions associated with a cough, dyspnea and, if the obstruction persists, cyanosis. A stridor, distinct from a wheeze, is often characteristic of tracheal obstructions, with the associated indrawing of the suprasternal notch, the epigastrium and the intercostal spaces. Tracheal obstructions due to mediastinal neoplasms not infrequently involve the esophagus as well, and consequently the patient complains of difficulty in swallowing and the regurgitation of food into the trachea due to esophageal overflow.

Auscultatory findings in tracheal obstructions are a loud stridor, wheeze and sonorous rales heard throughout the chest. These are generally accompanied by a diminution of the intensity and excursion of the breath sounds and a change in the percussion note, depending upon the degree of obstruction, i.e., if an obstructive emphysema has been produced, a hyperresonant or tympanitic note will be heard; whereas if the obstruction has produced a drowned lung, the chest will be dull to flat on percussion.

Obstructions at the bifurcation of the trachea are most interesting and many times extremely confusing because they may produce opposite types of obstruction in the two major bronchi. A tumor at the

bifurcation of the trachea may only partially obstruct both major bronchi, giving the findings of a bilateral obstructive emphysema. Or, it may completely occlude one bronchus and partially the other, resulting in an atelectasis of one lung with an obstructive emphysema of the other. Such patients at times present rapidly changing findings as one or the other major bronchus opens due to the extreme respiratory effort the patient makes to breathe. Two foreign bodies aspirated at the same time present findings identical to those of a tracheal obstruction. Thus, a child who chokes while eating peanuts may aspirate one part of a kernel into the right main bronchus, and then, following the next cough, inspire a second piece which is sucked into the left bronchus because of the occlusion of the right. The bilateral obstruction which results is indistinguishable from a tracheal obstruction.

OBSTRUCTION OF A MAJOR BRONCHUS

Partial obstruction of a major bronchus results in complete unilateral, obstructive emphysema. This is characterized by findings of limited expansion, relative hyperresonance to tympany and a marked suppression of breath sounds on the involved side. It may be accompanied by an inspiratory and expiratory wheeze and occasionally by rales or rhonchi. The heart and mediastinum are shifted to the uninvolved side on expiration, and, as Eloesser[21] states, the affected side of the chest is dilated rather than shrunken. With complete obstruction to the bronchus one may have the signs and symptoms of pneumonia or an empyema due to the "drowned lung." The distinguishing feature is the marked decrease in size of the lung distal to the point of obstruction compensated by a shift of the heart toward the involved side and an elevation of the diaphragm on the involved side. There are generally many rales present, but these depend to some extent upon the degree of secondary infection. There is a limitation of motion, dulness to flatness on percussion, and frequently marked bronchial breathing and bronchophony in the early stages of the atelectasis with breath sounds entirely absent if the atelectasis is of long duration or is extensive.

Obstruction of the bronchi leading to single lobes produces varying degrees of signs and symptoms dependent, generally, on the underlying pathological condition and degree of infection rather than upon the fact that the bronchus is obstructed. Both lower lobes are not infrequently involved in inflammatory obstruction,[29] giving relatively mild symptoms. However, the essential findings are similar to those described as following an obstruction of a major bronchus, except that they are more limited in their extent to the topographic outline of the lobe.

ROENTGEN FINDINGS

The roentgen aspects of the diagnosis of bronchial obstruction depend upon a complete roentgenographic study of the chest. The practice of basing an interpretation on one or two views of the chest leads to gross errors not only regarding the presence or absence of an obstruction, but also the location of the lesion. Fluroscopically, areas of density or emphysema, the motion of the diaphragms and the shifting position of the heart and mediastinum on inspiration and expiration are significant. While there are no actual roentgen findings in the bi-pass type of valve obstruction, unless the obstructing element itself is an opaque object, such as a common pin or a nail lying across the bronchial lumen, (see Fig. 8) the fluoroscopic findings of the next degree of bronchial obstruction, namely, that due to a check or ball-valve, are of greatest importance in establishing the diagnosis. They had been observed in individual cases for a number of years, but the extensive work of Manges[30] led to their widespread recognition and acceptance. He stated that the fluoroscopic findings of such an obstruction were an increase in the transparency of the affected lung, a depression and limitation of

motion of the diaphragm on the involved side, a displacement of the heart and mediastinal structures toward the uninvolved side on expiration, and finally a compensatory increase in the motion of the diaphragm on the uninvolved side. (Fig. 9.) Bronchoscopic observations have confirmed the roentgen interpretation that these findings were due to the presence of bronchial obstructions innumerable times. Further studies of these phenomena have been made to demonstrate areas of partial bronchial obstruction in which pulmonary tissue composing less than a lobe was involved. Golden[31] reported the fluoroscopic observation of abnormal respiratory displacement of the lower lung structures parallel with the movement of the diaphragm as the first evidence of obstructive emphysema in two cases.

As is well known, obstructive emphysema may be recorded on the roentgenogram by making exposures at extremes of the respiratory cycles and comparing the positions of the diaphragms and mediastinum as well as the density of the lungs on the two exposures. Thus, while a film made on deep inspiration shows both lungs completely inflated in this type of obstruction, the expiration film demonstrates the trapped air and its resulting physiologic phenomena as Manges described them.

The roentgen findings in complete bronchial obstruction are more obvious than those in partial obstruction because of the area of density distal to the obstruction. (Fig. 10.) In complete obstructions of the main bronchi, the well known findings are the shift of the heart and mediastinal structures toward the involved side during both phases of respiration, the elevation and fixation of the diaphragm on the involved side, and the density of the atelectatic lung. These findings are associated with a compensatory emphysema of the opposite side. Complete obstructions of the bronchi leading to single lobes or parts of lobes have less influence on the heart and mediastinal structures although they usually do give roentgen evidence of a shift of these structures toward the involved side, thus aiding in the differentiation between an atelectasis, a consolidation or a drowned lung. Atelectatic lobes or parts of lobes generally assume a more or less triangular shape and are frequently designated as triangular shadows.[32] It may be generally assumed that the bronchial obstruction in such triangular shadows lies at the apex of the triangle. However, it is essential

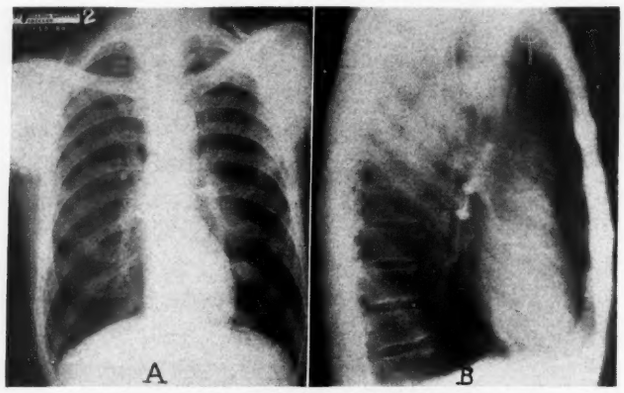

Fig. 8. Roentgenograms illustrating the first degree of bronchial obstruction. The patient was a bill-poster who aspirated two tacks when he fell off a ladder. A constant wheeze heard equally on both sides of the chest led to the diagnosis of "asthma." The foreign bodies, one in each main bronchus, were finally removed bronchoscopically eight years after the accident. Note the absence of pathologic changes in the lungs distal to the foreign bodies, characteristic of this type of bronchial obstruction.

that the shadow be studied roentgenographically in two planes, in order that the particular obstructed bronchus may be accurately localized.

BRONCHOGRAPHY

The importance of roentgen visualization of the bronchi was realized by Jackson[33] who in 1918 made the first satisfactory bronchograms by means of bismuth insufflations into the bronchi through a bronchoscope. Sicard and Forestier[34] using iodized poppy-seed oil (lipiodol) added a greatly improved radio-opaque medium which readily lent itself to intrabronchial instillation. At the present time, a variety of halogenated oils[35] is used, making the practice of roentgen visualization of the tracheobronchial tree a routine procedure in examinations of the bronchopulmonary system. A standard technic may be described as follows:[36] "In the adult the pharynx and larynx are anesthetized by means of 2 per cent pontocain spray followed by the instillation of 2 cc. of the same solution into and through the larynx, by means of a laryngeal syringe under mirror guidance. Again under mirror guidance a catheter on a curved wire stylette is passed through the larynx. The stylette is then removed as the catheter is advanced down the trachea. Under the fluoroscope the warmed oil is instilled into the desired area by manipulating the distal end of the catheter. In this manner the injection is limited to the area to be studied, and the subsequent films, taken immediately, present no confusion of superimposed shadows. Antero-posterior, lateral, and oblique films are indicated. In children, the catheter may be passed through the nostril and guided into the trachea under direct laryngoscopy. No anesthesia is necessary. Numerous bronchoscopists prefer the bronchoscope for the purpose of introducing the oil; some clinicians favor the perglottic method and others the laryngeal cannula, intubation cannula, or rubber catheter placed into the larynx as described above; some find that excellent results are obtainable by introducing the material supraglottically, while others prefer to inject the material through the cricothyroid membrane."

A detailed discussion of this subject with an extensive review of the literature and the technic may be obtained from the comprehensive article by Jackson and Bonnier[37] in which they stress the advantages of the catheter methods of instillation of the oil. From the roentgenographic standpoint they stress the importance of unilateral filling in certain cases in which the lateral view will play a determining factor in interpretation of the pathological condition, and the value of the oblique views if both major bronchi have been filled with oil.

Careful guidance of the oil into the areas requiring particular study is essential to obtain a complete visualization of the existing condition. This may be done only through fluoroscopic guidance of the oil as it enters the bronchi, and the tilting of the patient into such a position that the oil runs into the desired areas, whether they be lower, middle or upper lobes.

According to Farinas,[38] the use of iodized oil in bronchography has many disadvantages. These include its high viscosity which prevents its flow into certain stenotic areas, thus failing to show the extent of a stenosing lesion, as well as the fact that it remains in the lung parenchyma for long periods of time, especially in cases of incomplete bronchial stenosis. Farinas has substituted solutions of organic salts of iodine, such as uroselectan, for the iodized oil in such examinations. He finds these salts entirely nontoxic and nonirritating. Furthermore, their viscosity permits easy flow through any stenosis, and finally, they are rapidly and completely absorbed by the broncho-alveolar mucosa. Because of this rapid absorption it is necessary to take films while the solution is being injected.

Recent improvements in the technic of planography have added another diagnostic procedure which is available to aid

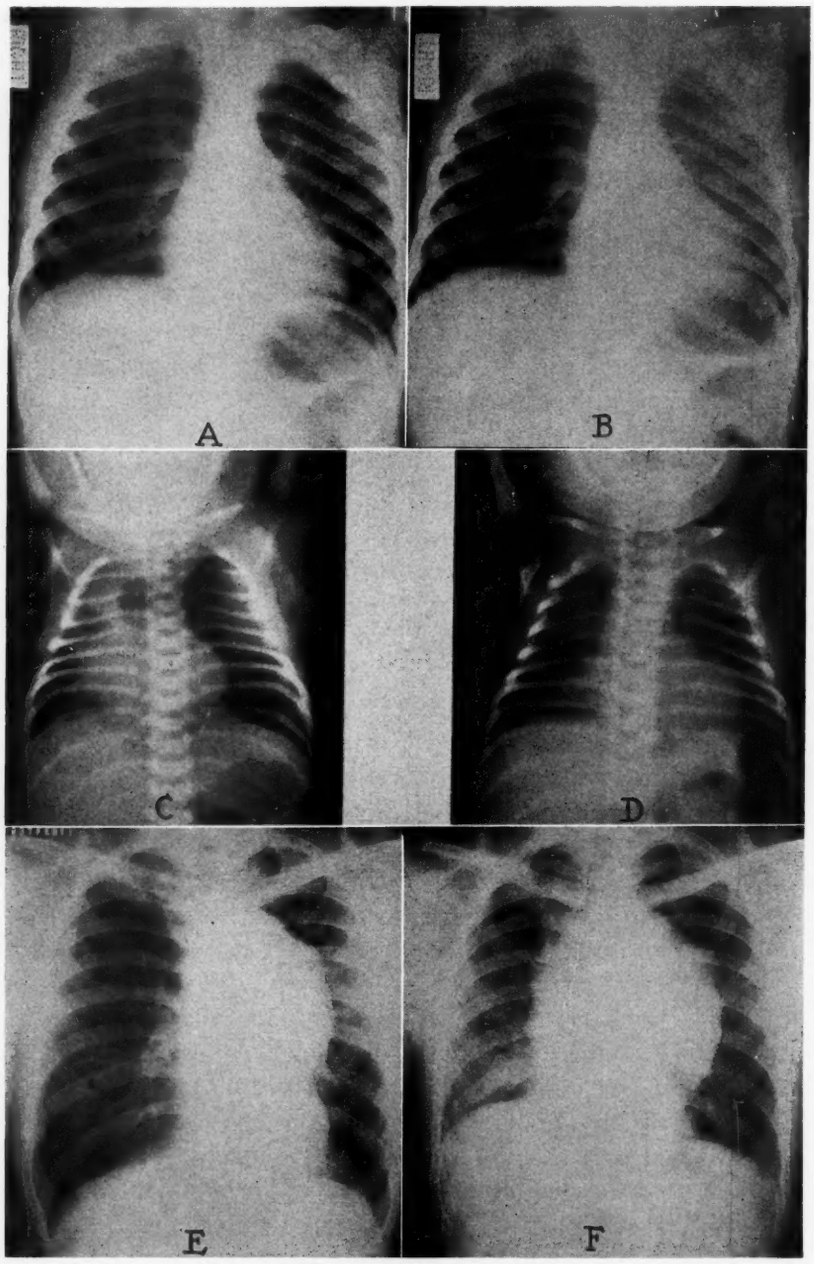

Fig. 9. Three cases of obstructive emphysema. A, inspiration, B, expiration films of a six-year-old girl who had an *intrabronchial* obstruction (peanut) in the right bronchus which functioned as a check valve. While a slight emphysema is apparent on the right side on inspiration, the diaphragms and position of the heart are normal. On expiration (B), however, the emphysema on the right side becomes very pronounced; the heart and mediastinum shift to the left and the right diaphragm remains stationary. The difference in the intercostal spaces on the two sides is striking and serves as an additional diagnostic feature. C, extreme obstructive emphysema of the left lung produced by an *endobronchial* lesion, a congenital web in the left main bronchus. The infant, twenty-four hours old, was in extreme respiratory distress, with tympany and absent breath sounds on the left side of the chest. A bronchoscopic examination revealed the web and it was dilated with forceps. D, roentgenogram forty-eight hours later. Breath sounds were equal on the two sides of the chest and fluoroscopy as well as the roentgenogram showed the heart and mediastinum to have returned to their normal positions. E, obstructive emphysema of the left lung due to an *extrabronchial* lesion, an aortic aneurism, which compressed the left main bronchus. Roentgenogram taken on deep inspiration. F, roentgenogram taken in deep expiration. The left diaphragm has remained stationary, the mediastinal structures have shifted toward the right and air has been trapped in the left lung.

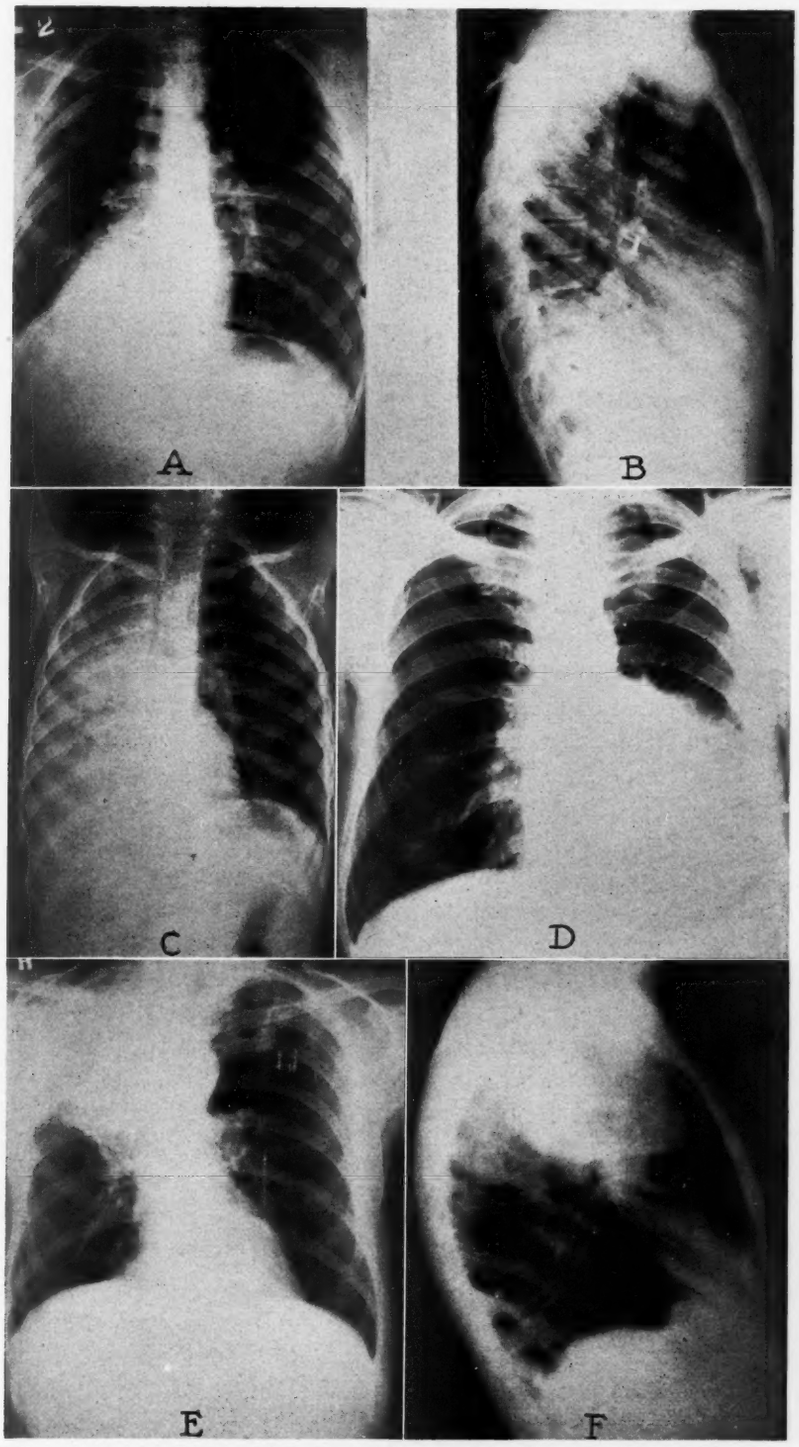

FIG. 10. Complete bronchial obstruction resulting in atelectasis. (A) anteroposterior and (B) right lateral views of a patient who aspirated a rivet. It is visualized in the right lower lobe bronchus and has caused a complete bronchial obstruction and atelectasis. The typical roentgen findings of atelectasis are illustrated by this and the other three cases shown. Note the density of the area distal to the obstruction, the elevation of the diaphragm on the involved side, and the shift of the mediastinal structures toward the involved side in each instance. C, four-year-old girl with atelectasis of the right lung due to a tuberculoma in the right main bronchus. D, atelectasis of the left lower lobe due to a bronchogenic carcinoma. E, atelectasis of the right upper lobe due to complete obstruction of the bronchus by a carcinoma. The configuration of the involved lobe is well illustrated in the lateral projection. F, histologic examination of tissue removed bronchoscopically established the diagnosis in each of the last three cases.

in establishing the presence of a bronchial obstruction.

BRONCHOSCOPY

In recent years bronchoscopy has become a universally accepted procedure. The bronchoscope has come to be considered a diagnostic instrument or speculum which permits direct inspection of the tracheobronchial tree of a patient of any age. Indications for bronchoscopy, which were formerly limited to the extraction of foreign bodies, have broadened so that now bronchoscopy is a routine aid in both the diagnosis and treatment of bronchial obstruction. Foreign bodies, tumors, bronchial compression and cicatricial stenoses produce identical physical and *x*-ray findings. Consequently, direct inspection must serve to establish the diagnosis of the actual obstructing agent. In the inflammatory diseases dried plaques of mucus, edematous infiltrations or the cicatrix produced by a healing ulcer may be responsible for the findings of obstruction and only the endoscopic view can establish the correct diagnosis. In cases of neoplastic diseases bronchoscopic inspection serves to establish the nature of the lesion through the removal of tissue for biopsy as well as to aid in determining its extent along the bronchial walls. In this respect, bronchoscopic inspection of the upper lobe bronchi and distal portions of the lower lobe bronchi deserves special consideration. Various technical measures are available to the bronchoscopist to make these examinations.[39] The use of retrograde telescopes and bronchoscopic mirrors serves to aid in the inspection of certain portions of the upper lobe bronchi. The use of the biplane fluoroscope to guide curved instruments into the upper lobe bronchi or into distal portions of the lower lobes facilitates the removal of tissue from those areas if they cannot be inspected directly. The use of artificial pneumothorax to bring an upper lobe bronchus into a more direct line with the main bronchus is of extreme value when an upper lobe bronchus is obstructed by a lesion suspected of being a neoplasm

DIFFERENTIAL DIAGNOSIS

Since a bronchial obstruction is dependent upon some underlying pathology due to a host of etiologic factors, the final diagnosis must be that of the underlying pathology. The differential diagnosis of bronchial obstruction, *per se*, consists of a consideration of those lesions simulating the effects of partial or complete obstruction. In the first category, bronchial asthma and pulmonary emphysema assume an important rôle because they both often closely simulate diseases due to bronchial obstruction. Possibly even more significant is the frequency with which a true bronchial obstruction is considered a simple asthma. The confusing factor most frequently is the wheeze produced by an obstruction which is asthmatic in character. It differs from an asthmatic wheeze, however, because the latter is usually altered at least temporarily on cough, and is expiratory in nature. The wheeze of bronchial obstruction is uninfluenced by cough and is inspiratory in character. In questionable cases, bronchoscopy and bronchography lead to a final diagnosis.

Lesions simulating the effects of complete bronchial obstruction or atelectasis are more numerous and consist essentially of pneumonic consolidation, empyema and tumors. Tumors large enough to give physical evidence of pulmonary pathological conditions may usually be recognized as such by their roentgen configuration. They are, of course, not infrequently involved in a process producing bronchial obstruction, when their differentiation from the atelectasis they have produced becomes almost impossible. Planography and overexposed roentgen films may aid in such differentiation, and again bronchoscopy and bronchography are indispensable.

Atelectasis is most frequently confused with intrapleural collections of fluid. A persistent atelectasis having had an acute

onset closely simulates a pneumonia followed by empyema. The actual differentiation depends on the position of the heart and mediastinum, fluid causing them to be pushed away from the involved side and atelectasis drawing them toward the affected side. The shifting dulness of fluid is another differentiating point. Since an empyema may follow an acute bronchial obstruction, however, the actual diagnosis may be made only after the fluid is withdrawn and the lung fails to expand.

The differentiation of an acute pneumonic consolidation from an acute atelectasis is usually dependent upon the history and the size of the involved lung in relation to its normal size. Postoperative massive collapse of the lung is frequently confused with postoperative pneumonia, but a careful physical and roentgen study of the position of the heart will be the most important distinguishing characteristic. Added difficulty is encountered, however, in cases of complete bronchial obstruction upon which is superimposed a severe suppurative infection to produce a "drowned lung". In this case the products of suppuration so completely fill the bronchi and lung distal to the obstruction that it retains its normal size and shape, yet has the physical and roentgen findings of consolidation. Differentiation in such a case may be made only through a history suggestive of bronchial obstruction, a failure of the pneumonic process to subside and the inspection of the involved bronchus with a bronchoscope.[40]

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FOREIGN BODIES IN THE LUNG

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FROM the viewpoint of thoracic surgery foreign bodies in the lung fall naturally into two very different classes: inspirated foreign body and penetrating foreign body. This is no quodlibetic distinction; the two classes are entirely different in almost every clinical feature.

PENETRATING FOREIGN BODY

In most instances foreign bodies entering the lung by any route other than the natural passages are projectiles. These cases usually call for care by the surgeon.

In case of small penetrating projectiles, however, if it is decided advisable to remove the intruder and there is no indication for opening the chest other than for removal, the foreign body can be removed through the mouth by bronchoscopy. The limit is the size of the foreign body; this limit is imposed by the size of the bronchus tributary to the invaded lobe.[2]

If the projectile has come to rest in a bronchus, removal is quite simple. If, however, the missile has lodged in parenchymal tissue, the biplane fluoroscope and penetrating forceps will be required at bronchoscopy. This procedure is almost always advisable when there are any indications that fabric of any kind has been carried in by the penetrating object, because in such cases suppuration is inevitable no matter how small the foreign body. Anyway, bronchoscopic removal is relatively such a minor procedure that no hesitation should be felt in advising it. Diagnostic bronchoscopy is advisable in all cases of projectile in the lung, no matter how large the intruder may be, to determine local conditions. Hemorrhage from a gunshot wound of one lung may be controlled by packing the tributary bronchus with bismuth subnitrate.[2]

INSPIRATED FOREIGN BODY IN THE LUNG

Foreign bodies reaching the lung by the natural passages constitute a class of case that has been, as stated by the late W. W. Keen, "entirely revolutionized by bronchoscopy." It may be added that the revolution concerns diagnosis as well as treatment. It is a platitude to say that the diagnosis of radiopaque foreign bodies has been revolutionized by the roentgen ray; but it may be added that the bronchoscopist and the radiologist in collaboration have made discoveries that have revolutionized the whole subject of diagnosis of pulmonary disease, including the interpretation of physical signs.[2,3,4] These matters concern chiefly the subject of bronchial obstruction which is covered by the article of Paul Holinger on other pages in this issue of the American Journal of Surgery.

TREATMENT OF FOREIGN BODY IN THE LUNG

All inspirated foreign bodies other than those minute particles concerned in pneumonoconiosis should be removed. In the days when removal of bronchially lodged foreign bodies involved thoracotomy there was ample ground for discussing advisability of removal; but in these days, when almost any localizable foreign body that has entered the lung by natural passages can be removed by the same route, it is unjustifiable to delay for encystment which is a remote possibility, or suppuration, which is a certainty.[2,4]

Emergency Cases. The most urgent and dangerous emergency occurring in cases of foreign body in the air passages is impending asphyxia. It therefore requires primary consideration.

Impending Asphyxia. When a foreign body, such as a bolus of meat or a large toy

is impacted in the laryngopharynx, or in the larynx itself, it not only obstructs the entrance of air, but unfortunately it permits escape of air past the foreign body on expiration. The effect of this one-way valvular action is that the normal thoracic movements are not only unable to draw in air, but *they actually pump air out of the lung.* The thus created bilateral atelectasis is so sudden and complete that it is always fatal, unless a physician happens to be present when the accident occurs.

The symptoms in such a case resemble those of an epileptic seizure in that the patient falls unconscious and is livid in color. The distinguishing features are (*a*) though the patient may clutch at his throat, there are no true convulsive movements of the extremities; (*b*) when unconsciousness supervenes the patient is motionless; and (*c*) there is indrawing at the guttural fossa.

When these three differential diagnostic points are observed the physician should not hesitate to do an emergency tracheotomy.[1] Artificial respiration will be necessary if respiratory movements have ceased. If the patient is intoxicated as is often the case, the artificial respiration may have to be kept up for a considerable time before the patient can be trusted to do his own breathing. In such cases oxygen, if promptly available, is invaluable. Still better in all cases of impending asphyxia is oxygen with carbon dioxide, 5 or 7 per cent admixture, a fact established by Yandell Henderson.

The emergency of impending asphyxia having been eliminated by tracheotomy the foreign body, such as meat impacted in the laryngopharynx, or a toy in the larynx, is then to be removed through natural passages, by direct laryngoscopy.

It might be thought that instead of doing the tracheotomy in case of pharyngeal impaction of such a foreign body as a large mass of meat, the physician could remove the obstructive mass with the finger and thus obviate a tracheotomy. This may be possible in some cases, but usually the efforts at digital removal increase the impaction, and the thus delayed tracheotomy may be too late.

Of course, if endoscopic means of removal are set up and ready a facile endoscopist could in a few seconds remove the obstruction, insert a bronchoscope through the mouth and resuscitate the patient by bronchoscopic insufflation of oxygen (plus carbon dioxide); but we are discussing here the emergencies that confront the practitioner. Every physician is, or should be able to do promptly a two-step, finger-guided emergency tracheotomy.[1,2]

The cardinal signs of obstructive laryngeal dyspnea calling for tracheotomy are as follows:[1,2] (1) Indrawing at the suprasternal notch; (2) indrawing around the clavicle; (3) indrawing of the intercostal spaces; (4) indrawing at the epigastrium forming a "funnel breast"; (5) restlessness; (6) choking and waking as soon as the aid of the voluntary respiratory muscles ceases in falling to sleep; (7) ashy color of the face, and (8) cyanosis is a dangerously late symptom.

Potential Asphyxia. A patient with a foreign body may be apparently perfectly normal yet be in danger of onset of asphyxia.

No consideration of the subject of emergencies in case of foreign body in the lung would be complete without a warning against the *symptomless interval.*[2] In our records are innumerable instances of a child dismissed with a negative opinion as to presence of a foreign body because of absence of symptoms at the time of examination. It does not seem to be realized generally that a foreign body can be lodged in a bronchus and yet the child be happy and playful, without cough or any obvious abnormality. Yet our clinical records show that in nearly 80 per cent of the cases of foreign body there was, after the initial gagging and choking, a symptomless interval varying in duration from a few hours in case of a bean or a peanut to a few months in case of a pin or needle. We have notes of many cases of a child dying of asphyxia on the way to our clinic. In a number of such

cases the child after an attack of choking and gagging on some foreign substance had been taken to a physician who had found "nothing wrong with the child." After a symptomless interval alarming symptoms developed and the patient died on the way to the hospital. The mechanism in such cases was of three kinds:

A. In some such cases an inspirated foreign body such as an orange seed was coughed loose and was impacted in the glottis.

B. In other cases a large foreign body, such as a half peanut kernel, turned crosswise at the bifurcation, obstructing ingress of air into both lungs; lifting on expiration it really acted as a one-way valve pumping air out of both lungs; asphyxia in such cases was sudden.

C. Quite frequently the asphyxia was due to increase in size of a tracheally or bronchially lodged foreign body, by its swelling, together with narrowing of the passages by mucosal swelling. This combination occurs always with dried beans and maize, and often with other bodies. These substances, swell enormously from absorption of water and, like all vegetal foreign bodies, cause a fulminating reaction in the tracheobronchial mucosa, the intensity being inversely as the age of the child. It takes very little swelling of the mucosa to narrow the airway in the small bronchi of children.[2,5]

In these cases of symptomless interval, the apparently normal, even playful behavior of the child gives no warning of the desperate impending asphyxia that may supervene suddenly a few hours later when the airway is occluded by the swelling of the foreign body and the mucosa. Of course, the greatest danger and urgency is in cases of foreign body in the trachea or at the bifurcation. Occlusion of one main bronchus does not cause dyspnea so long as the foreign body remains fixed. If, however, even in such a case the foreign body should be coughed loose, it is almost certain to be inspirated into and occlude the other main bronchus. Immediate asphyxia usually follows lodgment, because the previously obstructed lung being more or less atelectatic does not immediately resume function.[4]

BRONCHOSCOPY FOR FOREIGN BODY

In the foregoing we have considered the emergencies incidental to foreign body in the lung or obstructing the airway leading thereto. As a matter of fact, however, relatively few of the total number of cases of foreign body in the airway constitute emergencies.

Nonobstructive metallic foreign bodies such as needles, pins, small tacks, screws and nails in the tracheobronchial tree, do not constitute an emergency. The lungs are remarkably tolerant of such objects as long as they are not greatly obstructive. The barrier of the mucosa and bronchial wall is powerfully resistant to infection as long as ventilation and drainage are maintained.[5] In fact, iron and steel objects have an inhibitory effect on suppurative infections, probably by ionization. When there is obstruction to ventilation and drainage, however, suppuration will supervene. Even in such cases, there is no emergency. It will take weeks, in case of pins and needles months, for suppuration to get started, and even longer before the bronchial walls break down and abscess forms. There is, therefore, no justification for precipitate procedure. A well planned and carefully executed peroral bronchoscopic procedure will be successful in close to 100 per cent of the cases of inspirated foreign body.

Technic of Bronchoscopic Foreign Body Removal. The technic of insertion of the bronchoscope is easily acquired by competent instruction and training; the removal of a foreign body is, however, in many instances a highly technical, complicated procedure fraught with pitfalls and difficulties that may greatly endanger the patient's life. Adequate consideration of these technicalities is beyond the scope of a magazine article. They are fully considered in standard textbooks.

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The disturbances caused by abnormally situated thyroid lobes are dependent almost entirely upon the mechanical displacement of organs native to this region. . . . The organs involved are the trachea, the esophagus, the blood vessels, and the nerves.

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RECENT DEVELOPMENTS IN SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS

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INTRODUCTION

IN approaching the subject of recent developments in any field of surgical endeavor, it behooves one to review the historical background upon which rests the accepted philosophy of today. Of value are the records of past mistakes, that one may know what to avoid. Of even greater value, are the enduring truths that guide one into safer paths. Therefore, the historical approach, although of necessity in greatly condensed form, is employed in this discussion. Those who would delve further into this fascinating field should consult Murphy,[1] Sauerbruch,[2] Alexander,[3,4] Graham, Singer and Ballon,[5] and in particular, the writings and the discussions of Willy Meyer, which are replete with illuminative historical references, the accuracy of which cannot be questioned.

The discussion aims to trace the evolution of the philosophy of surgical treatment from the first serious efforts of the closing decades of the last century, through and including the thoughts expressed in the more important meetings of 1941. Details of technic, tabulations of indications and contraindications have, for the most part, been excluded. All those topics can be found readily in the literature. The viewpoint is that of a general surgeon; that of an individual.

Historically, three periods are recognized: the early period, the period of collapse therapy by trial and error and the period of rationalized collapse therapy. More or less arbitrary limits have been adopted. The first period embraces the last three decades of the nineteenth century. The second period embraces the first three decades of the present century. The third period dates from 1930.

Certain premises have been postulated, some purely surgical, others of broader implication:

First Premise. Rest under sound medical supervision, remains the unshaken cornerstone of treatment for pulmonary tuberculosis.

Second Premise. Pulmonary tuberculosis never was and never will be a "surgical disease." Operative intervention will prove beneficial to a fraction only, of all patients, and properly, therefore, is applicable to a fraction only.

Third Premise. The whole subject of surgical treatment of pulmonary tuberculosis is still in an evolutionary stage. Few, if any, "final words" have been written. There is vast room for improvement in every aspect of the work.

Fourth Premise. The practice of surgery includes the application of ten or more specific basic policies. Three of these basic policies are now, or have been at some time, utilized in the surgical treatment of pulmonary tuberculosis. They are: (1) the policy of total extirpation; (2) the policy of drainage, and (3) the policy of indirect surgery. Total extirpation is one of the most powerful weapons at the disposal of the surgeon. Drainage is likewise a powerful weapon. Indirect surgical attack is a very weak weapon, possibly surgery's weakest weapon.

EARLY PERIOD

Anesthesia, antisepsis and asepsis paved the way for the renaissance of surgery during the eighth decade of the nineteenth century. Experimental and clinical surgery advanced at a rapid pace. It was the day

of direct surgical attack: total extirpation, drainage, plastic operations, anastomoses, such similar procedures. The abdomen, the cranium, the thorax were all entered. The results of direct surgical attack for abdominal disease were definitely encouraging, and in that decade were laid the foundations of many a modern abdominal operation. Not so, however, in the case of thoracic disease, tuberculous or otherwise. It was soon discovered that the thorax could not be opened with impunity. "Open surgical pneumothorax," with its high rate of asphyxial death, became the deterring factor in intrathoracic surgery and remained so for years to come. Other than the Estlander and Schede operations for chronic empyema, which still survive in somewhat modified form, little if anything has come to us from the decade 1870 to 1880. It is significant that those operations were designed for patients whose mediastina had already become fixed by disease, and who could tolerate extensive operations upon their chest walls without the risk of paradoxical respiration and asphyxial death.

Lobectomy, open drainage, instillation of antiseptics into cavities, were all attempted on tuberculous patients. Sauerbruch,[2] at the 1908 International Tuberculosis Conference at Washington, D. C. referred to the results of these early operations as "worthless." One may fairly assume, in retrospect, that the early experiences were so discouraging that, in the treatment of pulmonary tuberculosis, all direct surgical attacks were abandoned and the general philosophy adopted that such procedures had no legitimate place in the treatment of the disease. This philosophy continued to dominate surgical and medical thought for more than fifty years.

Fortunately, however, the discouragements of the seventies did not deter all efforts. Direct surgical attack gave way to indirect methods. De Cérenville, Spengler, Quincke, Turban, Gourdet and Boiffin during the eighties and nineties were quietly persevering in the field of surgery. With the Estlander operation for chronic empyema as the prototype, and with a high mortality rate to combat, these early pioneers were groping toward the operation known as thoracoplasty. Forlanini, Potain and Murphy were developing therapeutic pneumothorax. Tuffier accomplished a successful extrapleural, transthoracic resection of a cavity-bearing apex and therewith laid the foundation for the pleurolytic operations. The dawn period of collapse therapy had arrived.

Just where the early experimental period ends and the established collapse therapy period begins, is hard to say. Let it be set arbitrarily at 1903, when Brauer, Friedrich and Sauerbruch all gathered at Marburg and commenced their monumental work on the development of thoracoplasty. One must not, however, assume that the philosophy of collapse therapy had, by that time, become widely accepted. It had not, and it is to be noted that in his Chairman's address before the Surgical Section of that 1908 Conference, C. H. Mayo,[6] keen and alert to surgical progress as he was, made not one reference to surgery as a therapeutic measure for pulmonary tuberculosis. Of forty-three papers on that Section program, only two (Robinson[7] and Sauerbruch[2]) dealt with thoracic surgery.

In rapid summary the results of the early period were:

Direct surgical attack (extirpation and drainage) had been tried, found wanting and discarded.

Therapeutic pneumothorax had been proved feasible. It was still in the experimental stage. It had not become widely accepted.

Multiple costectomy was in the experimental stage. Thoracoplasty, as it is known today, had hardly been visualized.

Parietal pleurolysis had been demonstrated as a method of surgical approach, and was being explored as a procedure of therapeutic value.

Only the hardiest pioneers were attempting any such therapeutic measures.

PERIOD OF COLLAPSE THERAPY BY TRIAL AND ERROR

At the beginning of this century therapeutic pneumothorax was the only procedure carried out in approximately the same manner as employed today. Thoracoplasty was known, but the operation of 1900 was utterly unlike the operation of 1941, or 1930, or 1920, or even 1910. Pleurolytic operations were occasionally performed. Anesthesia was crude. It did not seriously hamper progress in the abdomen, but it did hamper progress in thoracic work. It was definitely believed that direct surgery had no place in the treatment of pulmonary tuberculosis. Available statistics were meager and discouraging. There were no "trained thoracic surgeons." Only a few general surgeons and internists were seriously interested in exploring the possibilities of this field. Fascinating as it would be to trace the progress of this period in detail, time will permit only brief reference to some of the outstanding events.

It is doubtful if the phrase, "collapse therapy," was in use at the beginning of this period. It is certain that the concept of lung relaxation as recognized today, was not appreciated. Many still believed that resection of the upper ribs allowed the apex of the lung to "expand," become better ventilated and better vascularized. With such concepts, it is obvious that progress had to be achieved by the trial and error method.

The phrase, "collapse therapy," should include all measures, surgical, quasisurgical or mechanical, that will bring about diminution in volume of hemithorax or lung, with consequent physiological rest and increased prospect of healing of the tuberculous focus. "Collapse therapy" should not include drainage, total extirpation or other direct surgical procedures. As in descriptive zoology, so also in describing surgical operations, one may speak of genera, species and varieties. Extirpations or resections fall into one genus, drainage into another, collapse measures into still another genus.

The procedures of collapse therapy may be further divided into three species, depending upon the treatment of the pleural space, the respiratory motor mechanism or the chest wall proper. The last species must be further subdivided, depending on the treatment of the various anatomical components of the chest wall:

1. Diminution of the lung volume by separating the parietal and visceral pleural layers, but without crippling the respiratory motor mechanism or disturbing the chest wall structures. Therapeutic pneumothorax is the type variety of this species.

2. Diminution of lung volume by crippling the respiratory motor mechanism, without necessarily altering pleural or chest wall relationships. Diaphragmatic paralysis is the type variety of this species.

3. Diminution of lung volume by some anatomical alteration of the chest wall, but without necessarily crippling the respiratory motor mechanism or separating the pleural layers. The varieties are so numerous and divergent, that subspecies must be recognized.

3 *A. Diminution of lung volume by diminution of hemithorax volume, by removal of rigid chest wall structures.* Thoracoplasty is the type variety of this subspecies.

3 *B. Diminution of lung volume without diminution of hemithorax volume, by cleavage between chest wall structures regardless of resulting disturbance of blood supply.* The parietal pleurolytic operations are varieties of this subspecies.

3 *C. Miscellaneous limited operations on the chest wall.* A few highly diversified varieties such as subcostal compression, scaleniotomy, etc., have been reported.

By 1900, therapeutic pneumothorax had become a recognized procedure. Adhesions, then as now, defeated many a case. Open thoracotomy, for the section of adhesions, first reported by Friedrich in 1908, resulted in a high mortality rate from empyema. In 1912, Jacobaeus introduced intrapleural pneumonolysis, a much safer procedure.

One smiles when realizing that Jacobaeus turned the laparoscope into a thoracoscope, and very recently others have turned the thoracoscope back into a peritoneoscope. In 1922, Bernou introduced oleothorax.

Tuberculous empyema has been the by-product of therapeutic pneumothorax. Naturally, open drainage was first employed, but the results were not good and the pendulum swung far to the other extreme. Internists, surgeons, pathologists, bacteriologists, all contributed to the clarification of the subject. Alexander[8] has clearly stated the important considerations upon which sound treatment for each individual case may be formulated.

In 1911, Stuertz suggested phrenic nerve surgery. A wave of enthusiasm was followed by a wave of disappointment. In 1913, Friedrich performed intercostal neurectomy. In 1922, two modifications were introduced, phrenic exeresis and the radical dissection of the brachial plexus. Neither of these modifications added to the benefits to be expected. In 1933, Vadja reported pneumoperitoneum. Shot bags, braces, abdominal belts, lateral posture have all been suggested as mechanical adjuncts.

Operations involving chest wall structures early took two divergent paths, one leading toward thoracoplasty by multiple costectomy, the other toward the pleurolytic operations. In the thoracoplasty group, 1903 is a significant year. It marked the beginning of a systematic effort by Brauer, Friedrich and Sauerbruch, at Marburg, to evolve a safe and effective surgical operation for such patients as could not be treated successfully by pneumothorax. At that clinic pneumothorax was the procedure of choice and one is led to assume that only "pneumothorax failures" were referred to surgery. That may have been the basis for the tradition that pneumothorax must be tried and proved a failure before any other method may be attempted.

The experimental and clinical work at Marburg resulted in the operation known as Sauerbruch's extrapleural paravertebral thoracoplasty. For many years it was the standard operation but has now become obsolete. It served the very useful purpose of reducing operative mortality to a figure that encouraged, rather than discouraged further efforts in the field of tuberculosis surgery. Wilms also made important contributions. The principles underlying thoracoplasty were always surgically sound. The modifications that have appeared almost yearly have not altered the basic principles, but have added technical refinements and have incorporated the results of improved judgment.

The pleurolytic operations date back to 1891. Further developments hinged largely on the treatment of the "dead space." In 1901, Sarfert combined parietal pleurolysis with compression gauze packing. In 1910, Tuffier utilized fat plombage. In 1913, Bear employed paraffin. Other materials for plombage purposes, temporary or permanent, include muscle, rubber dam and rubber bags. Finally, air became the filling of choice. The pleurolytic operations have never been unanimously accepted as surgically sound. It is significant that they have waxed and waned in popularity.

The importance of anesthesia cannot be overemphasized. New agents, including the anesthetic gases, and great improvements in the technic of administration, have been significant factors in surgical progress.

It is characteristic that in the course of early surgical efforts, technic receives more attention than function or pathology. Mechanical execution excels judgment. Explanations for failure are sought first in methods of procedure, later in the laws of physiology and pathology. During the period of collapse therapy by trial and error there was a great accumulation of disjointed findings relating chiefly to technic. Years of careful study and correlation with the facts of physiology and pathology will be required to weld these discreet observations into one perfect composition. That task becomes the dominant theme of the period of rationalized therapy.

To refer to a period of trial and error does not mean that there were no great thinkers. Prophets are always with us. The prophets of one generation lay the foundation for the accepted work of the next generation. Not the prophets, but the rank and file determine the level of a given age.

In summary, the period of collapse therapy by trial and error saw: A world-wide awakening to the possibilities of surgical help for those suffering from pulmonary tuberculosis; a host of new operations introduced, some ephemeral, some of lasting value; great strides in technical improvement, but relatively little advance in precise diagnostic methods; extremely important improvements in the technic of anesthesia. General surgeons with special interest in thoracic work, co-operating with phthisiologists, were the clinical leaders of the period. Pathologists, physiologists and particularly workers in surgical research laboratories commenced an increasing volume of fundamental scientific investigation.

PERIOD OF RATIONALIZED COLLAPSE THERAPY

The beginning of rationalized collapse therapy may be arbitrarily placed at 1930. Two factors help in determining that date; the more important, the appearance of trained thoracic surgeons, the other, improvement in precise diagnostic methods and the continuation of research. The progress of the present period will be considered under the headings of training, diagnostic procedures, technical procedures, the optimum surgical environment and the effect on the philosophy of treatment.

Training. The pioneers who laid the foundation for thoracic surgery, including collapse therapy, were general surgeons and internists. They were not specially trained in thoracic problems. This was just as true of New World pioneers as of their Old World predecessors. Now special training in thoracic surgery can be secured. There are a limited number of approved residencies. Highly developed postgraduate training is offered by several universities in the United States and Canada. Eggers,[9] Graham,[10] Alexander[11] and Packard,[12] gave a clear pronouncement in 1936 that basic training in general surgery should precede all training in thoracic surgery. A committee of the American Association for Thoracic Surgery[13] further pronounced in 1937 that there is little likelihood that competent certification will be issued to the thoracic surgeon, other than certification in general surgery. This was reiterated in 1941.[14]

Year by year names are added to the list of men soundly trained in the fundamentals of surgery and the refinements of thoracic work. The effect of such a supply of competent surgeons can be predicated with a fair degree of accuracy: Sound logic will replace empiric effort; the demand for better diagnostic study will increase; the selection of cases for operation will be more intelligent; the quality of surgical care will improve; the variety of surgery will be broadened; a better grade of anesthesia will be demanded, and the environment in which the surgery is done will be improved.

Diagnostic Procedures. Diagnostic methods have been modified, during the last decade, in three very important particulars. These modifications are: the utilization of bronchoscopy, improvements in roentgenologic technic and the development of bronchospirometry. Other improvements have also been made.

Bronchoscopy, until approximately 1930, was regarded as definitely contraindicated by pulmonary tuberculosis; now it is not only permissible but usually desirable. Bronchoscopy has opened the way for the accurate study of the life history of tuberculous bronchitis. In connection with this very important subject one is referred to the writings of Barnwell, Littig and Culp,[15] Eloesser,[16] Samson and Culp,[17] Warren, Hammond and Tuttle,[18] Myerson,[19] Riggins,[20] and to the more recent symposium at the 1941 annual meeting of the

American Association for Thoracic Surgery by Chamberlain and Gordon,[21] Tuttle,[22] Alexander, Sommer and Ehler.[23] The gist of these contributions is to the effect that acute hyperplastic or ulcerative tuberculous bronchitis is a very serious complication, that it leads to bronchostenosis in a very high percentage of its victims and that bronchostenosis leads to severe secondary pulmonary suppuration. It has been demonstrated that the condition can be diagnosed and evidence is accumulating to show that all questionable cases should be bronchoscopically examined before instituting definitive treatment. Tuberculous laryngitis is, however, a valid contraindication to such examination.

Roentgenographic studies remain important. The recent development of tomography or laminography (let us hope that the arbiters of roentgenologic terminology will soon give us one acceptable term) is the most significant forward step.

Bronchospirometry or bronchospirography is the newest effort in precise diagnosis. Jacobaeus[24] gave an early report in 1937. Pinner, Leiner and Zavod[25] presented a scholarly review of this functional test in 1941. Some extraordinarily interesting observations have been made. The vast expanse of this field has not yet been glimpsed, but one may predict that the time will come when an estimate of respiratory function will be considered practical and important.

Sputum examination by refined methods, including identification of the tubercle bacillus by culture, has influenced the classification of surgical end results. It is no longer sufficient for sputum to be negative on direct smear; the day will arrive when it will no longer be sufficient for it to be negative on concentration; negative animal inoculations, negative cultures and negative gastric contents will be required.

Technical Procedures. Progress in technical procedures includes important modifications in thoracoplasty, air as a filling substance in pleurolytic operations, the reintroduction of drainage and extirpation operations and great improvement in anesthesia.

The extrafascial apicolysis of Semb is a distinct contribution. It is an anatomically precise dissection of all apical structures from their cervical and upper mediastinal relationships. It is surgically sound in that it is accomplished under direct visual control. It provides additional valuable caudad relaxation. But it adds to the hazards of thoracoplasty and therefore must be utilized judiciously. Gale and Oatway[26] have presented an excellent critical evaluation.

Aycock, Brantigan and Welch[27] have reported the clever utilization of a subpectoral air cushion in his so-called "extrafascial pneumothorax." It helps stabilize a flaccid chest wall, and adds an element of compression to the relaxation ordinarily obtained by thoracoplasty. Of necessity, it entails a large dead space which is an obvious disadvantage.

Proctor[28] has presented a careful study of "extrapleural pneumothorax," the newest variety of pleurolytic operation. Air is the filling substance, and air is well tolerated. One does not gain the impression, however, that this pleurolytic procedure will supplant the safer thoracoplasty.

Open drainage was early employed and rapidly fell into disrepute. In 1938, however, new interest was added to the subject by the report of Monaldi's closed drainage with gentle suction. Apparently drainage operations have returned upon a rationalized basis. There is much to make one pause and carefully reconsider the whole subject. One should consult Kupka and Bennett[29] for full details.

There is also the subject of palliative open drainage of the huge cavities with luxuriant growth of pyogenic organisms, by-products of tuberculous bronchostenosis. An authoritative pronouncement, based on a clear restatement of fundamental mechanical, physiologic and pathologic factors relative to the utilization of drainage is greatly needed. In the meantime it can only be said that the treatment of pulmonary tuberculosis will not be revolu-

tionized by drainage, and that one should proceed with the utmost caution.

Occasional case reports of pneumonectomy and lobectomy for pulmonary tuberculosis have appeared from time to time. The experiences of Dolley and Jones,[30] reported in 1940, provoked discussion that resulted in the tabulation of all available cases in the United States and Canada. The summary of the 1940 poll gave the following figures:

	Pneumonectomy	Lobectomy	Totals	
			No.	Per Cent
Apparently well	3	16	19	38
Improved	6	6	12	24
Unchanged	0	1	1	2
Worse	2	0	2	4
Dead	8	8	16	32
	19	31	50	100

Improvements in technic and in anesthesia have reduced mortality and have enabled 62 per cent of the patients to become arrested or improved. Operations of extirpation have, therefore, been reintroduced on a vastly sounder basis than sixty years ago. But the indications have not yet been clearly formulated.

Anesthesia, as an art and as a science, has advanced rapidly. The most important new agent is cyclopropane. The most significant achievement is the apnea technic. These improvements in anesthesia may prove to be the most important advances of the past decade.

Surgical Environment. Surgery, such as has been described, requires an environment that conforms in all respects to the established standards of American surgery. In particular, there must be assured: adequacy and competency of personnel, facilities for expert anesthesia, adequacy of equipment, facilities for expert surgical nursing, competent supporting laboratory and roentgenologic services and assurance that unhampered surgical judgment will control all clinical activities. This conformity to American surgical standards, upon which rests the moral right to maintain such a surgical program, is of paramount importance and supersedes all other considerations.

If such a surgical environment can be created within institutions primarily devoted to the care of the tuberculous patient, so much the better. Two great advantages, expert surgery and expert tuberculosis nursing and care, will thereby be secured for the patients. If such a surgical environment cannot be created within institutions devoted to the care of the tuberculous patient, then, on moral grounds, the patients should be transferred elsewhere for their surgical program.

Philosophy of Treatment. The generally accepted philosophy of treatment of pulmonary tuberculosis, if there ever has been such, has been built around the one word "rest." But instead of a coherent philosophy, one might prefer to recognize a series of independent concepts. Some of these concepts rest on a sound factual foundation and come to us with the weight of scientific truth. Other concepts rest on reasonable hypotheses and claim our respect, pending final verification or refutation. But some concepts appear to rest on tradition only and to be no more than blind formulae. The advent of men well trained in all the aspects of thoracic disease is certain to affect profoundly the philosophy of treatment. These trained men will challenge every concept of therapy. In their minds, the validity or nonvalidity of the concept will be determined, not by its venerability or legality, but by its conformity to basic natural law.

At present, two concepts remain unshaken: The first, that rest is the sheet anchor of treatment; the other, that proper institutional care is of inestimable advantage to the patient. All other concepts are being subjected to rigid scrutiny, and, one by one, must stand or fall on the basis of their own merit.

The concept that ether may not be used as an anesthetic has fallen. The concept that pulmonary tuberculosis is a valid contraindication to bronchoscopy has been refuted. The concept that direct surgery has no legitimate place in the treatment of pulmonary tuberculosis has been sharply challenged, if not actually refuted. The concept that therapeutic pneumothorax must be the first effort has been sharply challenged. Other old concepts have been questioned. New concepts are in process of formulation.

One hears more frequently the phrases, "reversible therapy" and "irreversible therapy," occasionally the phrases, "reversible pathology" and "irreversible pathology." It has long been accepted that "reversible pathology" should be treated by "reversible therapy." It has not been universally accepted that "irreversible pathology" should receive "irreversible therapy." However, a healthy doubt is rising in the minds of many as to whether a "reversible" therapeutic procedure has any logical place in the scheme of treatment of an "irreversible" lesion.

The general management of pleural complications has been quite thoroughly explored. Sound surgical recommendations have been formulated, but have not been universally accepted by other interests. There are still glaring examples of mismanagement.

It yet remains to formulate a sound therapeutic program for the unfortunate victims of tuberculous bronchitis, acute or chronic. More cases must be studied; more statistics must be compiled; more thought must be expended; more truths must be determined; more courage must be developed. Above all, sound clinical judgment must control the destinies of these unhappy people.

The surgeon looks at pneumothorax and thoracoplasty and thoughtfully reflects that they are the undisputed seniors in the ranks of collapse procedures. They have earned their right to a permanent place and one may confidently expect them to survive as long as surgery has a place in the treatment of pulmonary tuberculosis. They are alike in that they offer more effective collapse than any other measures; they are unlike in that one is reversible, the other irreversible; and further they are unlike in that one offers 70 to 75 per cent prospect of success and the other, today, offers 70 to 75 per cent prospect of failure.

The surgeon looks at the phrenic nerve operations and the pleurolytic operations and sees, at best, weak efforts, and, therefore, efforts to be used infrequently.

The surgeon looks at drainage operations and begins to see great possibilities, but also risks.

The surgeon looks at lobectomy and pneumonectomy and sees still greater possibilities, but also greater risks. He sees the need of rigid rules and requirements that would exclude all but 1 per cent or so of patients with pulmonary tuberculosis from such procedures. No extirpation operation should be done except with the moral certainty that the contemplated operation will remove all foci of active tuberculosis.

The surgeon looks at the whole field of therapy and sees evidence of sounder clinical judgment, better technical skill and more satisfying end results.

The older surgeon looks at his younger brother fresh from a thorough drilling in the basic sciences and says: "Yours is the task to sift and sift, to save the truth, to destroy the false, and to evolve a philosophy of treatment that will conform to natural and spiritual laws as they were created and have existed since the beginning of time."

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EMERGENCY TREATMENT OF PULMONARY HEMORRHAGE IN THE TUBERCULOUS PATIENT

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HEMOPTYSIS is a common symptom in pulmonary tuberculosis occurring in about 50 per cent of all cases at some time during the course of their disease. Various observers give figures varying from 20 to 80 per cent of cases, depending upon the stage of the disease, however, it is more common in the advanced cavernous type and usually is more extensive in this type of disease. It is the terminal event in a very small percentage of cases. About 1 or 2 per cent of patients will die as a direct and immediate result of a profuse pulmonary hemorrhage. It is extremely rare for a patient to die from the loss of blood *per se* but it is not uncommon for a patient to drown or suffocate in his own blood. The amount of bleeding also varies within wide limits from blood streaked sputum to massive hemorrhages of a quart or more, and it should be pointed out that the amount of blood expectorated is not an absolute index of the amount of bleeding that has occurred. There is almost always some blood retained in the lungs. In rare instances, more blood is retained in the lungs than is expelled.

In a general way there are three types of bleeding in pulmonary tuberculosis: When the blood occurs in streaks or as blood tinged sputum the cause is probably a capillary ooze from hyperemic areas or from the bronchial mucosa. The more common form is caused by an erosion or ulceration of a blood vessel by the actual tuberculous process. This is the usual cause in those cases in which we see from a teaspoonful to several ounces of pure blood expectorated, and is the type that may recur at varying intervals. Most profuse hemorrhages are brought about by destruction of lung tissue which leaves the blood vessel unsupported so that it ruptures, or aneurysmal dilitation occurs which later ruptures, giving rise to massive and at times, uncontrollable hemorrhages. There are other, rarer forms of bleeding that occur in tuberculosis without parenchymal disease. The most common of these are bleeding from ulceration of the bronchus and bleeding from the breakdown and ulceration of a hilum gland.

Hemoptysis is an alarming symptom both to the patient and to the physician; and even though it is rarely immediately fatal, it is often the beginning of a steady and rapid downhill course. In most instances, the after effects are more to be dreaded than the actual bleeding.

In the therapy of this symptom, there are a few fundamental principles to be followed: The first duties in treating this symptom are to put the patient at rest and do everything possible to allay his nervousness. Undoubtedly, nervousness and excitement on the part of the patient tend to increase the bleeding or to displace a blood clot that may be forming. Absolute rest should be enforced.

In approaching the treatment of this symptom, the first problem with which one is faced is the source of bleeding. In other words, has the patient been previously studied so that the most likely source of bleeding is known at the time that it occurs? Next, the therapeutic approach should vary somewhat with the extent of bleeding, namely, is it minimal, moderate or massive? In all pulmonary hemorrhages the patient should be put at absolute rest and reassured. Small hemorrhages usually stop of themselves without any treatment.

There is some divergence of opinion as to the position in which the patient should be placed. In my opinion, this is dependent, to a large extent, upon the above mentioned factors, namely, is the site of bleeding known, and second, is the extent of the bleeding slight or moderate, severe or massive? If the bleeding is slight to moderate and its source is not known, the patient should be placed in a semi-Fowler position. If the side of the bleeding is known, he should be turned on the bleeding side. If the bleeding is severe and the side is not known, and it cannot be determined, the patient should be placed flat on his abdomen with the foot of the bed elevated. If the side of the bleeding is known and the hemorrhage is severe or massive, the patient should be laid in the prone position, partially rotated with the bleeding side down. This will tend to splint the bleeding lung, will aid in postural drainage and will prevent aspiration of blood and bacilli laden sputum to the contralateral lung.

Mild sedation is indicated in practically every case. Sedatives should be used to the extent of quieting the cough but not to stop it and to allay the nervousness and excitement of the patient. Small doses of codeine, such as ¼ gr. every three hours in conjunction with the bromides and barbiturates are usually adequate. The stronger opiates, if used at all, should be used in very small doses. They reduce the cough reflex to such a point that they predispose to the retention of blood in the lungs with a subsequent development of atelectasis and tuberculous or pyogenic bronchopneumonia.

After the institution of absolute rest and mild sedation, the next problem to be faced is, can the bleeding be controlled medically or should surgical or mechanical means be resorted to? Medical treatment, at best, is not very satisfactory. The number of remedies which have been suggested and used, many of them with diametrically opposite action, will indicate that we have no certain methods of arresting the bleeding from the lungs, and, also, that the medical profession, realizing its impotence, is still ready to grasp any straw which offers any hope of success.

The various drugs that have been recommended fall into two main groups: those which affect the circulation and those which increase the coagulability of the blood. In spite of the enthusiastic reports of many, most of these drugs are of little or no value; however, some of these should be mentioned. Atropine sulfate is thought by some to be of considerable benefit and may be given in doses of $\frac{1}{100}$ gr. (0.6 mg.) hypodermically. The nitrites have been recommended because they lower the systemic blood pressure and in that way may lower the blood pressure in the pulmonary circulation. They may be given in the form of amyl nitrite, nitroglycerin or sodium nitrite. Ergot was formerly used to a large extent but it actually causes a rise in pulmonary blood pressure and for this reason cannot be recommended. Emetine is considered by some to be almost a specific in moderate or even severe types of pulmonary hemorrhage. It may be given in the form of emetine hydrochloride in 1 gr. (0.065 Gm.) doses hypodermically. Beneficial effects may be due to dilitation of the splanchnic vessels with reduction in the pulmonary blood pressure.

Drugs which purportedly influence the coagulability of the blood, have been tried extensively. Calcium in the form of calcium lactate or gluconate by mouth has been used for many years, but it has no demonstrable effect on bleeding unless given in massive doses. It has been highly recommended as a placebo that does no harm. Calcium gluconate given intravenously, is thought by some to be of value, and it is suggested that viosterol be administered at the same time to make certain of increasing the blood calcium, which is essential to the process of coagulation.

Coagulants, such as fibrogen, thromboplastin, etc., are of value in reducing the coagulation time and may be of some benefit in treatment, provided it has been shown by laboratory tests that altered coagulation is a factor in the bleeding.

The intravenous use of Congo red has been highly recommended as a treatment for hemoptysis. Ten cc. of a 1 per cent solution given daily for several doses has been recommended. It is supposed to reduce the clotting time, increase the blood platelets and the blood fibrin content. In my somewhat limited experience, it has been of little value; and when one considers the pathologic condition of tuberculous hemoptysis, that is, bleeding from a blood vessel with infiltrated and degenerative walls, it is easy to see how little can be expected from a hemostatic. The use of various vitamins has been suggested particularly vitamin C and vitamin K. They are of no proved value unless there is a know vitamin deficiency. Snake venom has been recommended in recurring hemoptysis but, in my opinion, there is little justification for its use.

In contradistinction to the above measures, the mechanical means of controlling pulmonary hemorrhage offer the double advantage of controlling the bleeding as well as the underlying disease. Of the various procedures, pneumothorax is the one of choice. In considering collapse therapy for pulmonary hemorrhage, it is, of course, necessary to know from which side the hemorrhage is coming. This may be very difficult as the tuberculous lesion may be bilateral and may possibly have open cavities in both lungs as likely sources of bleeding. Physical signs are of value in determining which side is bleeding, but may on rare occasions be misleading due to aspirated blood. Quite often the patient can tell, with fair accuracy, the side from which the blood is coming. This type of case should be approached with great caution. It has been my practice to put off attempting pneumothorax until I could be relatively sure as to the source of bleeding. Hemoptysis, in my opinion, is the one indication for rapid induction of pneumothorax. By this method 500 to 1,000 cc. of air are instilled at the initial treatment and a refill is given within twelve to twenty-four hours and thereafter as often as needed, dependent upon the control of the hemorrhage and the type of collapse obtained. In my opinion, except in rare instances, positive pressure should not be given on the initial treatment. Theoretically, pneumothorax is the ideal treatment for pulmonary hemorrhage; but from a practical viewpoint, a satisfactory collapse frequently cannot be induced due either to the absence of free pleural space or the presence of extensive adhesions, which prevent collapse of that portion of the lung from which the bleeding is coming. With prompt recognition of this complication by early thoracoscopic examination, one can occasionally convert an unsatisfactory pneumothorax into a satisfactory one. This procedure may appear radical, but in my experience has given good results in a few cases in which it was resorted to.

In cases in which there is no free pleural space, phrenic nerve interruption has been of value in at least temporarily controlling hemorrhages. In a few instances we have supplemented phrenic nerve interruption with pneumoperitoneum, with satisfactory results. There are some cases of recurring pulmonary hemorrhage in which pneumothorax and phrenic interruption have failed to control the bleeding; in these instances thoracoplasty is indicated. This procedure is justified only in those cases in which the bleeding is reasonably well controlled on rest, but recurs on the slightest activity. It is understood, of course, that the condition of the contralateral lung justifies thoracoplasty. The results from thoracoplasty are usually quite satisfactory. However, there are a number of cases in the literature in which thoracoplasty failed to control recurring hemopthsis in which lobectomy was done with some good results. It has been suggested that in this type of case ligation of the branch of the pulmonary artery to the affected lobe is the procedure of choice.

The type of supportive treatment in pulmonary hemorrhage depends to a large extent upon the degree of the hemorrhage Absolute rest is the most important factor. During the stage of active bleeding, the

patient should be watched continuously. If it becomes evident that considerable blood is being retained in the lungs, prompt and vigorous postural drainage may be life saving. We have seen patients saved by the attendant's hanging the patient's head down over the edge of the bed. In a general way, food and fluid should be restricted for at least twenty-four hours. Soft, easily digested food may be given, avoiding stimulants and hot drinks. The diet should be kept relatively dry for several days thereafter. Attention to the bowels should never be neglected, and it is important that the patient not be allowed to strain at stool for fear of causing a recurrence of bleeding. It has been my practice to wait for twenty-four hours and then give magnesium sulfate in a quantity sufficient to produce easy movement of the bowels.

If the patient has lost considerable blood and signs of shock are present, small transfusions of plasma or whole blood are indicated. This will be necessary only in rare instances.

For after-care or convalescent treatment, the patient should be kept rigidly in bed from a week to ten days after a hemoptysis, during which time he should be thoroughly checked to be sure that there has been no progression of his disease before gradually returning to his previous routine treatment for his disease.

CONCLUSIONS

The most important factors in the emergency treatment of pulmonary hemorrhage are: (1) Rigid bed rest, the position in bed dependent upon the extent and source of bleeding; (2) reassurance of the patient; (3) mild sedation; (4) if the above methods fail, or the hemoptyses recur, collapse therapy should be considered, and of the various methods, pneumothorax is the procedure of choice. This failing, the more radical procedures may be resorted to.

TEMPORARY VERSUS PERMANENT PARALYSIS OF THE DIAPHRAGM IN THE TREATMENT OF TUBERCULOSIS OF THE LUNGS

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IT is perhaps inevitable that a certain aura of mystery should surround the operation of phrenic nerve interruption. In the first place, up to comparatively recent times it was believed that an active diaphragm was essential for the vital function of respiration. Secondly, the causing of the paralysis of a muscle as a therapeutic measure is unique in the realm of surgery. Moreover, this muscle is probably the most active one in the body next to the heart muscle. It has important and diverse functions in the respiratory and circulatory mechanisms as well as providing the dividing barrier between the thorax and the abdomen. Thirdly, the operation as we have come to know it, when well performed, is one of the neatest in its execution and most dramatic and far-reaching in its results in all the surgical repertoire. Through a tiny incision in the neck a small nerve trunk is exposed which on suffering interruption of its axons results in paralysis of half of the great contracting partition of the midtrunk. At the beginning of the present century, Schroeder and Green in animal experiments showed that first one hemidiaphragm and then both sides of the muscle could be put out of action by bilateral phrenicectomy without serious impairment of breathing. Gradually, phrenic interruption came to be recognized as a valuable therapeutic measure in certain diseases of the lungs, notably in pulmonary tuberculosis. Like most newly discovered operative procedures it suffered from overvaluation at first, and at one period it was being performed in almost mass production style in various centers for the treatment of tuberculosis of the lungs. In more recent years the early surge of enthusiasm has died down and with better recognition of the indications resulting from experience gained by observing the many thousands of patients who have now been operated upon, the operation is, or should be, taking its proper place in the general armamentarium of collapse therapy. One says "should be" advisedly because even today there is quite a large amount of divergence of opinion concerning certain aspects of the operation, even among phthisiologists and thoracic surgeons. For the general surgeon who is often called upon to do a phrenic nerve operation, the most important question next to "when should one paralyze the hemidiaphragm in the treatment of pulmonary tuberculosis?" is, "should one cause permanent or temporary paralysis in this or that particular case?" It must be emphasized at once that with very rare exceptions one should always do a temporary paralysis at first. One can never say with certainty beforehand what advantages or disadvantages may accrue as a result of the operation. Should the desired result be obtained, the temporary paralysis can always be easily repeated or the paralysis can be made permanent. If, on the other hand, the operation should fail to influence the disease to the extent required, or if certain disconcerting complications should be the direct result, then no permanent harm will have been done because the diaphragm will once again regain its full function in due course.

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INDICATIONS

The modern concept envisages two distinct views of the operation: (1) The phrenic nerve operation used as an independent therapeutic measure unassociated with other forms of collapse therapy and aided only by the sanatorium régime. (2) The phrenic nerve operation used as (a) a preparatory measure, and (b) an auxiliary measure to some other form of collapse therapy.

INDEPENDENT PHRENIC NERVE OPERATION

As regards this use, John Alexander has pointed out the great guiding principle of the indications. The operation of phrenic interruption used as an independent measure is very effective in early and limited lesions, very ineffective in old and extensive lesions, and notoriously unpredictable in lesions intermediate in type between these two extremes. This is true irrespective of the position of the lesion in the lung field; and those who have had experience with the use of the operation combined with bed rest and sanatorium régime in the treatment of the early lesion, particularly the early uncavitated subclavicular infiltration, are convinced of its great value in this type of case. Most of these patients will do well on bed rest and sanatorium régime alone, but the time required to effect a cure will be shorter and the eventual cure surer in those patients who have had the additional benefit of a diaphragmatic paralysis. In the patient with the early uncavitated lesion or in whom the cavity as seen on the *x*-ray plate is very minute (1 to 1½ cm. in diameter, for example), there will at first be no question of a permanent paralysis. Six to twelve months' sanatorium régime combined with temporary paralysis repeated once or twice will be sufficient to bring about arrest in most instances. The second or third period of temporary paralysis will usually coincide with the period when the patient will have returned home and resumed his work and normal activities. If in an occasional case at the end of this period after the diaphragm has regained its function there should appear once more signs of reactivation of the disease, it might fairly be judged that a permanently idle diaphragm will be necessary to keep the disease controlled, and a lasting paralysis may be instituted or an artificial pneumothorax may be tried.

In an early lesion which shows signs of cavitation greater in extent than that mentioned above, it is probably wiser to induce pneumothorax in the beginning than to hope for lasting results from phrenic interruption. Experience with pneumothorax therapy has shown that in order to achieve permanent closure of tubercular cavities the collapse must be maintained for a period of at least two and one-half years. Such a term of collapse from diaphragmatic paralysis would necessitate either a permanent interruption or the repeating of the temporary phrenic interruption on three or four occasions. After the operation has been repeated on two occasions, it becomes extremely difficult technically to perform again a fourth time because of the scar tissue that forms and the displacement of the familiar landmarks. Moreover, the injury sustained by the phrenic nerve at each succeeding operation makes return of function less and less likely the more often the operation has been done. In this way one will end up with a permanently paralyzed diaphragm to treat a lesion which could be controlled in all probability more completely and certainly more quickly by an artificial pneumothorax, with much less permanent loss of respiratory function. To those who will point out the objection that pneumothorax treatment is a tedious business which, in addition, lays the patient open to such serious complications as empyema and spontaneous pneumothorax, one may reply that a complete pneumothorax is still the most perfect and efficient form of collapse therapy known to us, and that in the type of case which we are considering, one rarely meets with complications if the pneumothorax is instituted and conducted properly. This argument holds chiefly for lesions

in the upper portion of the lung. When the lesion is in the lower part of the lung, however, one may more readily and with much less misgiving do a permanent paralysis, once the complete effectiveness of the measure has been proved after one or two terms of temporary paralysis. In these cases one loses little in the way of respiratory function because the lower part of the lung is already partially destroyed by the disease, and it will be an advantage to keep it permanently at rest. The function of the healthy upper part of the lung will not be greatly affected by the paralysis of the diaphragm; whereas in treating disease of the upper lobe by permanent paralysis, one loses forever in large part the function of the lower healthy part of the lung.

Apart from the type of case just discussed, the operation is being less and less performed as an independent measure, and according to O'Shaugnessy is being used as such only by those who have a limited repertoire of collapse operations at their disposal. A far more important use of the procedure is first as a preparatory measure, and secondly as an auxiliary measure to some other collapse maneuver.

PREPARATORY PHRENIC NERVE INTERRUPTION

When the operation is intended as a preparatory procedure, the paralysis will invariably be a temporary one. As such, it is used in preparation for (1) artificial pneumothorax and (2) extrapleural thoracoplasty.

1. Kremer strongly advises its use before the attempt at induction of an artificial pneumothorax in cases of fresh, predominantly exudative disease, especially when cavitation lying near the surface of the lung is present. The rest and relaxation resulting from paralysis of the diaphragm will react favorably on these acute lesions causing the exudative reaction to become absorbed and the temperature to become lowered. In this way the pleural exudates rapidly turning to empyemas, and the spontaneous pneumothoraces due to cavity rupture (which are seen unfortunately only too often when these patients are treated by early pneumothorax) are avoided.

2. The second important use of phrenic nerve interruption as a preparatory measure, as advocated by Hein, is in the case of a lesion extensive enough to warrant a thoracoplasty but which is too unstable to permit the performance of this operation at once. Often in this type of case one is confronted with a small recent lesion in the lung of the opposite side which requires some form of active treatment before one dares to contemplate the undertaking of an extensive collapse operation on the more diseased side. A wise plan of campaign in such a case is to induce a protective, so-called mantle, pneumothorax on the less diseased side, and the induction is usually successful in the case of a lung which is the site of such limited disease. Experience has shown that it is desirable to postpone the contemplated thoracoplasty until the contralateral pneumothorax is well established. This will usually take a period of some two to three months to accomplish, and during this time the more diseased side will be made much more suitable for thoracoplasty as the result of the beneficial effect of diaphragmatic paralysis on that side. Seeing that most authorities are now of the opinion that it is safer to have the diaphragm functioning on the operative side during and immediately after the performance of a thoracoplasty so as to avoid lower lobe atelectasis and retention of secretions, one is well advised to cause phrenic interruption by the freezing technic after which the diaphragmatic function is apt to return in the course of two to four months.

AUXILIARY PHRENIC NERVE INTERRUPTION

As an auxiliary measure, phrenic nerve interruption may be used to supplement (1) artificial pneumothorax, (2) extrapleural thoracoplasty and (3) extrapleural pneumonolysis.

1. *Phrenic Nerve Interruption Used as an Auxiliary to Artificial Pneumothorax.* In this connection perhaps its most valuable use is in the case in which artificial pneumothorax has succeeded in partially closing a

cavity or cavities but in which complete closure is prevented by pleural adhesions. The anchoring of the lung to the parietes by adhesions in one direction, together with the intermittent tugging action of the diaphragm in another direction subjects the diseased part to continual irritation and does not permit complete apposition and firm healing of the cavity walls. This may be true even though the actual lumen appears obliterated roentgenologically. Division of the adhesions by the method of Jacobeus is the first and most obvious thing to try. In those cases in which this does not prove feasible, two courses of action present themselves. Either the pneumothorax may be abandoned and a thoracoplasty performed, or paralysis of the diaphragm may be tried. The latter is, of course, much the simpler and less dangerous procedure; and the relaxation of the adhesions which it provides will in many instances permit satisfactory cavity closure and eventual healing. Dumarest has made a particularly interesting study of the results of phrenic nerve interruption as used in conjunction with artificial pneumothorax. His diagrams best show the types of cases in which favorable and unfavorable results may be anticipated. As one would expect, those cases in which the lower lobe has already retracted upward from the diaphragm and those in which the cavity lies in a part of the lung which is adherent to the parietes on all sides present the least favorable prognosis. (Fig. 1.)

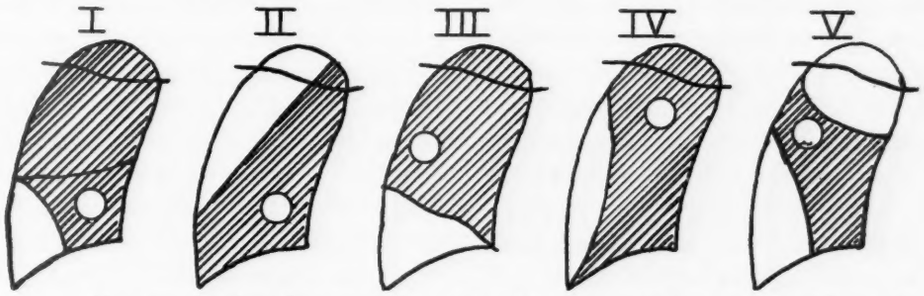

Fig. 1. Diagram showing the types of cases in which favorable and unfavorable results may be anticipated. A favorable result may be expected from phrenic interruption in examples I, II and V. In IV the result is doubtful and in III no success may be anticipated.

These cases are no exception to the almost general rule that the initial interruption should be a temporary one. Since the artificial pneumothorax should be maintained over a period of at least two and one-half years after roentgenological and clinical evidence of cavity closure has been reached, and inasumuch as a temporary interruption would have to be repeated on four or more occasions so as to keep the cavity walls constantly apposed and the lung in a more or less fixed position thereby giving it the best chance to heal, it is logical to hold that after the effectiveness of the measure has been proved, one should in these cases go ahead and do a permanent interruption before the diaphragm regains its full function again. In many instances this can and should be done. In cases with bilateral disease, however, and especially in cases in which the contralateral lesion is as yet unstable, one should never rush in to do a permanent paralysis. The contralateral lesion is apt to progress at any time to such an extent that collapse measures may be needed to control it and the permanent reduction in respiratory function resulting from a phrenicectomy may make the performance of the necessary operation extremely hazardous or even impossible. This has been the fate of many patients with bilateral disease on whom permanent phrenic nerve interruptions have been done at an early stage in their disease, and the outcome, of course, has only too often been a fatal one. In such cases one should continue to repeat the temporary paralysis only as often as it seems absolutely necessary and proves feasible; and a seasoned judgment resulting from a long experience

in dealing with the behavior of tubercular lesions is necessary to determine the best course of action in each individual case.

The other important use of phrenic interruption in connection with pneumothorax therapy is in the patient whose disease has been satisfactorily controlled by a pneumothorax which has been maintained over a number of years and which is now being abandoned intentionally or is being lost because of obliterative pleuritis, and in whom such extensive destruction of lung tissue has occurred as to make the eventual full expansion of the lung to fill its original hemithorax hazardous and inconvenient. The hazard arises from the strain to which the quiescent lesion would be subjected under such circumstances, and the inconvenience would derive from the unpleasant subjective phenomena associated with high degrees of negative pressure in the thorax. In extreme instances this inconvenience may grow to real danger when the heart and other mediastinal organs are put under such tension that serious circulatory embarrassment may result. Permanent paralysis of the hemidiaphragm will cause a considerable reduction in the size of the hemithorax, and will help to circumvent the dangers outlined above. Some writers go so far as to advise paralyzing the diaphragm temporarily during the critical re-expansion phase in every pneumothorax, claiming that in this way the frequently observed exudate ex vacuo is not encountered and that the re-expansion is a smoother and less abrupt process. The type of pleural exudate that accompanies re-expansion is, however, usually harmless and it is generally agreed that in most instances phrenic nerve interruption is unnecessary in this group.

2. *Phrenic Nerve Interruption Used as an Auxiliary to Extrapleural Thoracoplasty.* Diaphragmatic paralysis used in conjunction with thoracoplasty has its chief value in those cases showing disease involving practically the whole of the lung in question. The modern thoracoplasty is very effective in controlling upper and midlung disease, but is less efficient in influencing lesions situated in the lower one-third. Such disease processes lying as they do over the broad floor of the thorax, and exposed to the continual disturbing influence of the contracting diaphragm are less subject to the relaxation and compression from above and from the circumference of the thorax, such as is provided by thoracoplasty. Paralysis of the diaphragm which is best done after the completion of the thoracoplasty will give the necessary relaxation from below. Provided the other lung is capable of carrying on the respiratory function adequately, one will desire permanent exclusion of the totally diseased side from activity, and in such a case one may well do a permanent interruption of the phrenic at once.

3. *Phrenic Nerve Interruption Used as an Auxiliary to Extrapleural Pneumonolysis.* Some time after the establishment of an extrapleural pneumothorax or oleothorax it occasionally happens that signs of residual cavitation are detectable just beneath the floor of the artificially created extrapleural space. A temporary paralysis of the diaphragm is always well worth trying in such a case before one decides to resort to thoracoplasty. In this way one subjects the cavity to a collapse effect acting from above and below.

Phrenic nerve interruption is of value in treating certain other aspects of pulmonary tuberculosis in what may be classified as a miscellaneous group:

a. Hemoptysis. Some authorities, including John Alexander, are firm in their advocacy of the operation in the treatment of hemoptysis met with in pulmonary tuberculosis. Others discount its value in this instance and hold that it may even be harmful in that it may lead to stagnation of tubercular infected blood in the lung thereby causing atelectasis and dissemination of the tuberculosis. The fact remains that the bleeding does become controlled in many instances after performing the operation. One must be sure from which lung the blood is coming and the respiratory functional capacity must be known to be adequate. Then if an attempt at

induction of pneumothorax has failed, one is justified in doing a temporary paralysis of the diaphragm on the side in question.

b. Spontaneous Pneumothorax. In some instances of spontaneous pneumothorax, the aperture in the visceral pleura is held open by pleural adhesions and in this way the healing of the rent and absorption of the air is prevented. Relaxation of the adhesions and closure of the tear will in occasional cases be made possible by temporarily paralyzing the diaphragm.

c. Tubercular Empyema. In tubercular empyemas that necessitate thoracoplasty to effect closure of the empyema cavity, ascent of the paralyzed diaphragm will occasionally help in obliterating the defect. Provided the respiratory functional capacity warrants it, and seeing that the tubercular empyema cavity is somewhat analogous to a giant pulmonary cavity for which one requires permanent collapse, one may well do permanent interruption in these cases at once.

TECHNIC

The exposure of the phrenic nerve is best done through a transverse incision lying about one finger's breadth above and parallel to the clavicle, its medial end being placed just internal to the lateral border of the clavicular head of the sternocleido mastoid muscle, and extending laterally for a distance of about three inches. The operation can admittedly be done through an incision half this length, but taking into consideration the important and even vital structures that are found in the field and allowing for that cardinal requisite of good surgery—good exposure—one need make no apology for an adequate incision. Skin closure by means of Michel clips, removable in forty-eight to seventy-two hours, leaves a linear scar which becomes well nigh indetectable in the course of time.

Premedication in the form of 1½ gr. of nembutal two hours before the operation, followed by ⅙ gr. of morphine sulfate and 1/100 gr. of atropine sulfate one hour before, usually makes the procedure more comfortable for the patient. The best posture is the supine position with the face turned away from the side of the operation. The skin is prepared according to the wishes of the surgeon and the operative site is adequately draped. Local anesthesia (0.5 per cent novocain) is the anesthesia of choice in the adult, though cyclopropane or nitrous oxide anesthesia may be required for an unco-operative child.

Exposure of the Nerve. The incision is carried down through the skin, superficial fascia and platysma, and superficial layer of the deep cervical fascia, with the scalpel, until one exposes the supraclavicular fat pad. The sternocleido mastoid muscle with the underlying carotid sheath is retracted medially and the external jugular vein is retracted laterally.

The fat pad is divided by repeatedly inserting and opening the tip of a small Metzenbaum scissors and presently one comes down on the prevertebral fascia covering the scaleni muscles. Superficial to this the posterior belly of the omohyoid makes its appearance and as it is usually somewhat sensitive it is injected with a couple of cubic centimeters of novocain and is then retracted upwards. Below the omohyoid the transverse cervical vessels are seen coursing laterally and are carefully avoided.

The phrenic nerve is found lying deep to the prevertebral fascia on the superficial surface of the anterior scalene muscle and gradually edging inward toward the medial border of this muscle as it descends. Lateral to the anterior scalene and emerging from the depression between it and the middle scalene one sees the brachial plexus. Extreme care is exercised in handling the plexus. The phrenic nerve is freed from the prevertebral fascia with a special nerve hook and its identity is proved by lightly pinching it with a hemostat when a convulsive twitching of the patient's epigastrium is caused by the resulting contraction of the hemidiaphragm. One-half to 1 cm. of novocain is then injected into the nerve sheath and the nerve is left for the present. A very careful search is now made for the accessory phrenic nerve or nerves, in the

angle between the brachial plexus and the main phrenic trunk. This is the most usual position for the accessory nerves and the search should be carried up to the point of emergence of the fifth cervical nerve from underneath the lateral border of the anterior scalene and down to the clavicle. It must be stated unequivocably that no phrenic interruption is satisfactorily performed without the finding and resection of any accessory phrenic nerves present. Medial to the main trunk and around the inner border of the scalenus anticus one looks for a possible second phrenic nerve, because cases with double phrenic trunks at this level have been reported. Such a second trunk, if found, must be treated similarly to an accessory phrenic nerve. Finally, seeing that an accessory phrenic nerve occasionally runs in the same sheath as the nerve to the subclavius muscle, this latter nerve which arises from Erbs point of junction of the fifth and sixth cervical nerves and passes down anterior to the third part of the subclavian artery and the subclavian vein is resected in those cases in which it is found. Any strands which appear to be accessories are tested in a similar fashion to the main trunk by pinching with a hemostat and watching for an epigastric twitch.

From now on the course to be pursued depends on whether temporary or permanent paralysis of the hemidiaphragm is desired.

TREATMENT OF THE NERVE

1. *Temporary Interruption:* (*a*) *Crushing.* The phrenic trunk is crushed three or four times in succession at exactly the same place with a hemostat. Too vigorous a crushing or a crushing involving too extensive a length of the nerve may cause permanent paralysis. Several centimeters of any accessory phrenics found are resected. This technic will give a paralysis of approximately six months in duration.

(*b*) *Freezing.* A strip of gauze about 2 cm. in breadth is laid crosswise under the phrenic trunk. One or 2 cm. of the nerve's length is then frozen with an ethyl chloride spray or carbon dioxide snow; the accessories or their equivalents are treated as in the crushing technic. Freezing will cause interruption for a period of two to four months.

2. *Permanent Interruption.* Although avulsing the phrenic has been and still is being used widely to cause permanent paralysis of the diaphragm, the radical phrenicectomy as proposed by Goetze and modified by J. Alexander is much the safer operation. In this procedure 2 to 4 cm. of the main phrenic trunk, together with one or more centimeters of all accessory roots, are resected.

In closing, the superficial fascia is brought together with two or three fine sutures of silk or catgut. Approximation of the skin edges is effected with Michel clips and a dry gauze dressing is applied.

COMPLICATIONS OF PHRENIC INTERRUPTION

1. *Immediate Operative Complications:* (a) Severe hemorrhage may occur from injury to a normal or abnormal blood vessel found in the region of the operation, notably from the normal ascending or transverse cervical arteries or from an abnormal branch of the subclavian artery. On the left side the thoracic duct is liable to injury. (b) Pulmonary air embolism is a possibility, due to injury to the internal or external jugular or subclavian veins. This accident while possibly of minor significance in people with healthy lungs is apt to cause serious respiratory insufficiency when the lungs are partially destroyed by disease. (c) Injury to the vagus, sympathetic trunk, or even parts of the brachial plexus has been known to occur.

2. *Remote Complications:* (a) Dyspnea is likely to occur in patients with markedly lowered respiratory capacity. One should bear this possibility in mind in particular in elderly patients, over fifty years of age who have a tendency to emphysema, and in patients of any age with extensive destruction of lung tissue. (b) Undue displacement of the abdominal organs may occur. The

gastrocardial complex has been observed when unusually high ascent of the diaphragm on the left side interferes with the normal mechanism of the cardia and leads to air swallowing and permanent distention of the stomach. An uncomfortable feeling of fullness and intractable vomiting is the result.

Mechanical ileus has been reported after right-sided operations due to displacement of the duodenum, and constipation as the result of interposition of the large bowel between the liver and the diaphragm. These latter complications are only extremely rarely observed and probably then only in cases with pre-existing abdominal abnormalities such as adhesions or ptoses. When suspicion of such possibilities exists, as for example in cases with peptic ulcer, one should be slow to perform phrenic interruption and one should never undertake an initial permanent interruption.

(c) Undue displacement of the heart and great vessels may result. The healthy cardiovascular system is not likely to be seriously disturbed by paralysis of the hemidiaphragm. Embarrassment may occur, however, in cases with cardiovascular disease, and experiments by Nissen and Wustmann on the caval blood flow are of interest in that they suggest the detrimental effects of permanent diaphragmatic paralysis on the cardiovascular system when the latter begins to be taxed by the common degenerative changes accompanying advancing years.

POSTOPERATIVE TREATMENT

Strict attention to the after-care is one of the most important aspects of phrenic nerve interruption, and one upon which the degree of success or failure of the measure often depends. It is remarkable what a pronounced rise of the diaphragm occurs in patients who are kept strictly on bed rest for long periods after the operation for some such reason as Potts' disease of the spine. If the greatest benefit is to be secured, absolutely strict bed rest should be enforced for at least three months postoperatively. In addition, the foot of the bed should be raised ten to fifteen inches and the patient should lie as much as possible on the side of operation. In this way, the weight of the abdominal viscera pressing continually upward against the paralyzed diaphragm and the minimal ventilation of the lung resulting from strict bed rest will cause a striking ascent of the diaphragm in those cases in which it is not fixed by adhesions. The position is naturally uncomfortable at first; but if the raising of the foot of the bed is brought about gradually in steps of three to four inches at a time, a co-operative patient will quickly become accustomed to it. The comparatively recent work by Kremer and Von der Weth with the Roentgen-Kymogram shows some promise that by means of this technic one can predict with fair accuracy the amount of success or failure to be expected from a phrenic nerve interruption in treating an upper lobe lesion of more than minimal extent.

SUMMARY

The operation of phrenic nerve interruption is of the nature of a minor procedure in an operative field whose near-vital anatomical structures makes the possibility of serious complications a dangerous trap for the unwary operator. The absolute necessity of strict attention to the indications is borne out by the fact that its well proved efficacy in suitable cases is balanced by its inefficacy and grave dangers in unsuitable cases. The wise surgeon must think twice before he puts half of a patient's diaphragm forever out of action, and the initial paralysis should hardly ever be a permanent one.

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THE RECOGNITION AND MANAGEMENT OF BRONCHOPLEURAL FISTULA

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A BRONCHOPLEURAL fistula is a communication between the pleural cavity and the bronchial tree, however tortuous the tract may be. It may occur as a complication of a neglected pneumococcic or other pyogenic empyema, as a result of drainage of a lung abscess, or as a result of trauma, but is most commonly found in pulmonary tuberculosis, frequently as a complication of artificial pneumothorax. This paper will concern itself particularly with the problem as encountered in tuberculosis.

The fistula may be of any size, from microscopic to several centimeters in diameter. It may be transient and may be open constantly or intermittently.

The significance of a fistula lies not in the communication itself but in its complications. These fall into three groups, any or all of which may obtain in a given case: (1) Alteration in the intrapleural pressure; (2) infection of the pleural cavity by either or both (a) the tubercle bacillus (from the rupture of a caseous subpleural focus or the sloughing of a cavity wall); (b) pyogenic organisms; and (3) aspiration of pleural exudate into a bronchus. Symptomatically annoying, this frequently causes extensive spread of the disease.

Incidence. To Coryllos and his coworkers[5,6,22] must go the credit for making us realize the high incidence of this condition. Until gas analyses were used, only those fistulas were diagnosed which were large enough to cause abnormal pressure readings or to allow expectoration of appreciable quantities of pleural fluid, either with or without the admixture of a dye such as methylene blue or gentian violet.

In Table 1 is given the incidence of this complication as listed by various authors. The average is about 5 per cent of artificial pneumothorax cases but represents only those clinically recognized. If we include the small or intermittent leaks which are found only by gas analysis, the figure is much higher. Auerbach and Lipstein[2] give an incidence of 7.8 per cent in 1,000 autopsies of those dying of pulmonary tuberculosis. Of this 7.8 per cent about one-fifth were found in previously uncollapsed lungs. It should be borne in mind that even at autopsy some fistulas, particularly small ones, will escape detection, so the true incidence would probably be in excess of the figures found.

TABLE 1
INCIDENCE OF BRONCHOPLEURAL FISTULA IN PNEUMOTHORAX

Author	Year	Material	Perforation	Per Cent
Burrell*.....	....	171 cases pneumothorax	4	2.3
Matson[20].....	1935	480 cases pneumothorax	16	3.3
Weisman[29].....	1936	150 cases pneumothorax	..	3.3
		72 cases effusion	..	7.0
Dumarest and Brette*.....	....	265 cases pneumothorax	11	4.1
Burnard*.....	....	300 cases pneumothorax	14	4.6
Fishberg[12] (Montefiore Hospital)..	1932	.....	..	5.0
Andrew Peters*..	....	.....	..	5.0
Nicklas, Franklin and Zavod.[25]	1937	375 cases pneumothorax	19	5.0
		253 cases effusion	..	7.5
Forlanini*.....	....	139 cases pneumothorax	8	5.7
Auerbach and Lipstein[2].....	1939	1000 autopsies	78	7.8
Brown and Hayes[16]	1933	230 cases pneumothorax	..	10.0

* Quoted by Fishberg.

Bronchopleural fistula is more prone to occur in the early months of pneumothorax, particularly if the collapse is incomplete, but it may occur where a successful pneumothorax has been maintained for a long time. In the series of Brown and Hayes,[16] the average time for occurrence of the fistula in twenty-three cases was the twenty-ninth

month after initiation of pneumothorax. This is probably later than most series would give because in seven of these cases it occurred after the fortieth month and in one after six and one-half years.

Auerbach and Lipstein[2] found at autopsy that those perforations which occurred within six months of pneumothorax were usually associated with a fulminating caseous pneumonia, whereas those fistulas occurring from one to four years later were associated with chronic pulmonary tuberculosis.

Relationship of Empyema to Fistula. If we consider the relationship of empyema to fistula, we find it is a very close one. Alexander[1] states, "It is probable that a majority of all tuberculous empyemas, certainly those secondarily infected, started from a tiny, unrecognized perforation or a larger rupture of the lung." Coryllos,[6] on the basis of his studies of pleural gas analysis, states that bronchial fistula is the most important factor in the pathogenesis of purulent effusion or the change of clear effusion to purulent. However, without gas analysis, this is difficult to prove, although, as Hayes[16] points out, it should be suspected in every case of pleurisy with severe onset even if it cannot be proved at the time.

Without using gas analysis, fistula can be proved in about one-third of all tuberculous empyemas. Skavlem and his co-workers[27] found 20.5 per cent; Woodruff,[30] 30.5 per cent; Nicklas, Franklin and Zavod,[25] 35.1 per cent; Brown and Hayes,[16] 38 per cent; Mainin,[19] 41.3 per cent. Conversely, however, where there is a gross bronchopleural fistula, tuberculous or mixed infection empyema is almost universally found.

Nicklas, Franklin and Zavod's nineteen cases of bronchopleural fistula were discovered among their fifty-four cases of empyema; probably in the majority of instances the empyema was due to the fistula. Auerbach and Lipstein[2] state that in their seventy-eight cases of fistula discovered at autopsy, empyema was present in every instance with the exception of five cases in which the fistulas developed too near death for fluid to develop.

While the majority of large bronchial fistulas are associated with a tuberculous empyema, it is not always easy to prove a secondary pyogenic infection. Many authors have failed to find pyogenic bacteria in a large number of tuberculous fluids in which a fistula is known to be present. It would seem that many of these failures are due to an insufficient number of examinations over a long enough period of time. However, Harpöth and Gad[14] in nine successive cases of fistula over a period of two years failed to find secondary organisms in a single instance. Nicklas and his co-workers[25] found secondaries in only six of nineteen known perforations. In the Saranac Lake series collected by the present author,[30] there were forty-seven cases of fistula and in twelve no secondaries were isolated. However, these twelve included instances in which the patients died quickly after the perforation and other instances in which there were very few bacteriological examinations. In discussing that series Rogers[26] stated that he had had a similar experience—seven negatives in twenty empyemas. Auerbach and Lipstein[2] in their autopsy series found that in forty-four of their cases in which the bacteriology was accurately known, mixed infection was present in twenty-seven, or 60 per cent.

Size and Location. Fistulas range from a microscopic perforation to the actual sloughing of a cavity wall, in which case the fistula may measure several centimeters in diameter. Inasmuch as these fistulas are due to sloughing of tuberculous foci, they are found where the tuberculosis is most extensive and where cavities and adhesions are most likely to be, namely, in the apex of the lung. Auerbach and Lipstein[2] reported 73 per cent in the upper lobe. The minute ones which can be diagnosed only by gas analysis or by variations in intrapleural pressures are the most numerous.

Symptoms. There is no one symptom complex which will cover all cases of fistula. First, it should be borne in mind that, while

the majority of fistulas occur in cases of therapeutic pneumothorax, they can occur in previously uncollapsed lungs. In such a case a fistula initiates the pneumothorax.

Frequently the formation of a fistula is attended by violent constitutional symptoms. In other instances there is no dramatic change and without the aid of gas

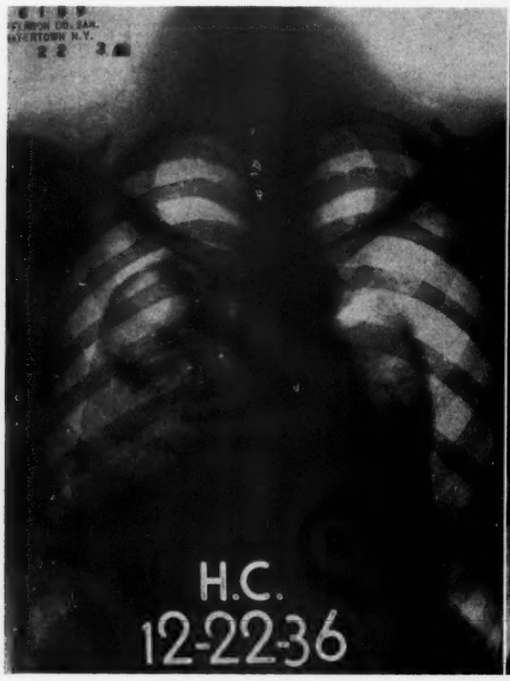

Fig. 1.

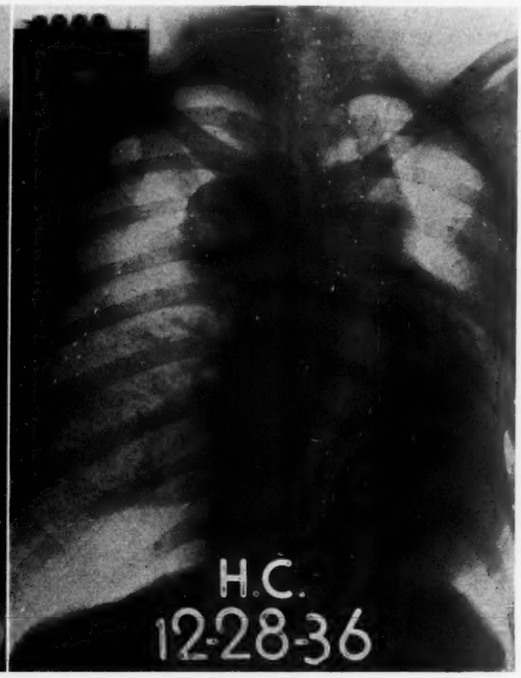

Fig. 2.

Figs. 1 and 2. Case 1. Bilateral pneumothorax before and after development of bronchopleural fistula on the right with tension pneumothorax.

If a fistula is small and temporary, there may be no symptoms and many such cases escape detection.

Dyspnea, if not the most common symptom, is one of the most distressing and one which attracts the attention of both the patient and the physician.

Pain in the chest, sudden fever, unexplained malaise, the expectoration of material either greater in amount or differing in character from that previously raised, are all symptoms, which, when they occur in tuberculosis, should suggest to the clinician the possibility of bronchopleural fistula. Any of these occurring in the presence of an artificial pneumothorax should make him strongly suspect it. In addition, the rapid formation of fluid, particularly if it is cloudy, or the change of fluid already present from clear to cloudy, or a collapse greater in amount than would seem likely from the amount of air injected are all suggestive of the presence of a fistula.

analysis the presence of many fistulas would never be suspected, much less proved.

Diagnosis. In some cases the diagnosis of fistula can be made on the basis of symptoms and signs; in others, the symptoms are merely suggestive and other tests are necessary.

There are two symptom complexes which are easily recognized clinically and which, when present, are pathognomonic: The first is due to a marked change in the intrapleural pressure, with or without signs of infection, and the second is the expectoration of pleural fluid. Both of these are discussed subsequently.

Case 1. Illustrating the first type of small fistula with developing tension pneumothorax is the case of Mrs. H. C., who was under the care of Dr. S. E. Simpson. (Figs. 1 and 2.)

The author had performed a closed intrapleural pneumonolysis on the right side eleven days before the film of December 22, 1936.

Convalescence was uneventful until the afternoon of the twenty-second when she developed increasing dyspnea, and between the time of the first film and the one of the twenty-eighth, 4,700 cc. of air had been withdrawn from the right side and 800 cc. of air from the left.

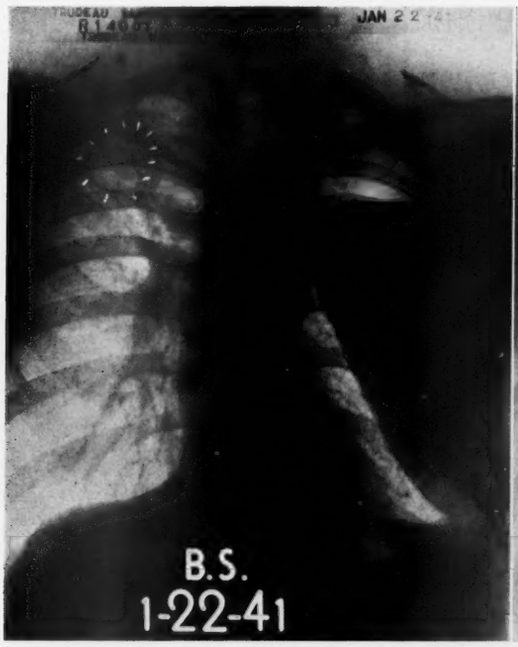

Fig. 3.

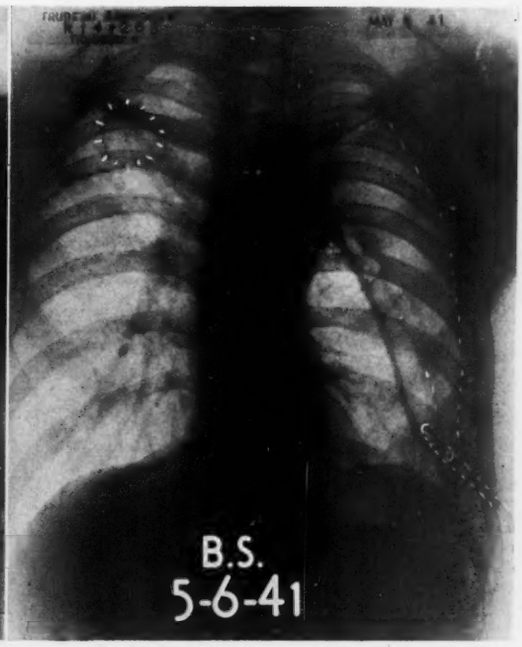

Fig. 4.

Figs. 3 and 4. Case II. Bilateral pulmonary tuberculosis with tuberculous empyema on the left. Figure 3 shows the size of the pocket and Figure 4 shows the position of the catheter. The technic of the insertion of this catheter is illustrated in Figure 9.

Case II. The second commonly recognized type, in which a patient suddenly expectorates pleural fluid, is illustrated by the Case of Mr. B. S., who was under the care of Dr. J. Woods Price. (Figs. 3 and 4.) Fluid had been forming slowly and on February 6, 1941, 850 cc. were aspirated. During the aspiration fluid was replaced by an equal amount of air. The patient was left with a pressure of +3 + 1, which was quite usual. Following aspiration there was increased elevation of temperature for four days. However, the course was otherwise uneventful until the night of February 14, when he began to cough and raise fluid when recumbent. This fluid was thinner and had a different taste from the sputum which he had previously raised. He soon found that the cough stopped on sitting up or when lying on the left side. This case will be discussed further under "Treatment."

In Cases I and II, and many more like them, the diagnosis is obvious. They represent the minority of fistulas. Many of the others are more difficult to diagnose and are proved only by other diagnostic methods, at postmortem or not at all. In the autopsy series collected by Auerbach and Lipstein[2] in only 67 per cent was the diagnosis made antemortem.

Whenever fistula is suspected we have a number of criteria and tests at our command. Frequently only one will be positive; in other instances, several. For purposes of discussion we shall list them separately: (1) Physical examination, (2) expectoration of fluid or dyes, (3) pressure changes in pneumothorax space, (4) gas analysis under varying conditions, (5) *x*-ray or fluoroscopy, and (6) thoracoscopy.

Physical Examination. There is no constant physical finding in all bronchopleural fistulas, although, as Barnwell[3] brings out, "Any respiratory difficulty appearing in a pneumothorax patient should be regarded as due to pulmonary perforation until proved otherwise by a determination of the intrapleural pressure." As he further points out, the symptoms of acute perforation are entirely respiratory until pressure on the

great veins and heart result in circulatory disturbance and until general anoxemia produces convulsions. If the examiner can hear the bubbling of air through fluid or a metallic tinkle, it is probably pathognomonic of a fistula. This is seldom present. If it can be demonstrated that there is a shift of the mediastinum with displacement of the heart away from the suspected side, fistula is strongly suggested. Before it can be considered as absolute proof, one has to be certain that the shift is a new development unrelated to an excessive amount of pneumothorax refill or the development of a large pleural effusion. There may be extreme fullness of the affected side of the hemithorax with bulging of the interspaces. There may be rapid respiration, evidence of air hunger and cyanosis. All of these signs are suggestive; none is pathognomonic. The most important point on physical examination is to recognize dyspnea when present and to realize that increasing dyspnea presents an acute emergency. When it is present, the immediate treatment is directed toward its control. For instance, in Case 1 air was first withdrawn at 3 A.M. on December 23. Despite the appearance of the film of December 28, no further air was withdrawn because dyspnea was not marked.

Expectoration of Fluid or Dyes. Commonly, a patient who has had fluid in the pleural cavity will begin to expectorate larger amounts or material of a different character than previously. This is characteristically influenced by the patient's posture. In other words, when the orifice is above the fluid level, he will not expectorate fluid; when it is below the fluid level, he probably will. This was true in Case 11. Dye placed in the pleural cavity and recognized in the sputum is a great aid when in doubt. Gentian violet when used as an irrigating solution is an excellent indicator. It was used in this instance and seen in the expectoration several days later. Methylene blue is a commonly used indicator, 10 cc. of a 1 per cent solution being used. It tends to fade over a period of days in the presence of pus. The taste of gomenol is characteristic in those cases who have oleothorax. All of these are proof positive when spat up, but the failure to expectorate them does not mean that there is no communication. In the series of Auerbach and Lipstein,[2] previously referred to, out of thirty-four instances in which fistula was proved at postmortem and in which methylene blue was injected during life, it was expectorated in only twenty-two instances, or in two-thirds of the cases. Nor is the failure to expectorate dye due always to the fact that a fistula is small. It may occur when there is a large opening if the opening is kept above the fluid level. In other instances, if a fistula has been present but is sealed over by fibrin or temporarily closed by pressure, neither fluid nor dye may be spat up immediately.

Aromatic oils, such as wintergreen, have been used as indicators, usually a few drops being placed on a cotton filter in the line of a pneumothorax machine. This is less sensitive than the dye, is open to many errors of interpretation and has little practical value.

Pressure Changes in Pneumothorax Space. These changes may be of two types: (*a*) Where the fistula is so large and so patent that no matter how much air is injected into the pleural cavity or withdrawn from it, no change in intrapleural pressure can be obtained. In such an instance the diagnosis is clear; but, if there is any doubt, methylene blue placed in the pleural space would probably be expectorated promptly, although it might be necessary to tilt the patient in various positions until one is found in which the perforation would be beneath the fluid level. (*b*) Where the opening is very small or intermittent, there is a tendency to build up a positive pressure or to fail to maintain a highly negative pressure if one is produced.

This is well illustrated by the following summary of a patient who was under the care of Dr. John Hayes at Sanatorium Gabriels:

Case III, Mr. C. C. Tuberculous empyema, right side, aspiration and irrigation, with attempt to re-expand, using high negative pressure.

Date	Pressure in Cm. of H_2O		Amount in Cc. Injected or Aspirated	Remarks
	Before Treating	After Treating		
6–25–38	−5−0	−23−12	75	Seropurulent material aspirated; washed with 550 cc. saline
6–28–38	−6−2	−18−12	350	Air aspirated
7–1–38	−9−12	−24−12	50	Purulent reddish-colored fluid aspirated; washed with 300 cc. saline
7–5–38	−6−2	−15−10	250	Air aspirated
7–8–38	−2+2	−26−16	50	Brownish pus aspirated; washed with 200 cc. saline
7–12–38	+6+8	−24−18	450	Air aspirated; no fluid obtainable; washed with 500 cc. saline; added 50 cc. gentian violet, 1:1000
7–15–38	+4+8	−12−2		
7–21–38	−0+4	−4+2	100	Gentian-colored pus aspirated; washed with 300 cc. saline; introduction of saline into pleural cavity initiated spasm of coughing with raising of gentian violet

It will be noted that on July 8, 1938 the initial pressure was $-2 + 2$, when three days previously the final pressure was $-15 - 10$. This reading in itself was quite suggestive of perforation but more marked was the initial reading four days later of $+6 + 8$. In the absence of a marked increase in fluid, that was presumptive evidence of perforation. It was thirteen days after the first suspicious pressure reading before the diagnosis was verified by the raising of sputum and dye. Dye had been in the pleural cavity for six days before it was expectorated.

The pneumothorax record of Case I is here given in detail as it illustrates an intermittently open fistula. It is also an instance of control of symptoms by repeated aspiration of large amounts of air.

Case I, Mrs. H. C. Bilateral pneumothorax. Pneumonolysis, right, 12–9–36 and 12–11–36.

It will be noticed that pneumothorax pressures through December 22, 1936 were what might be expected. However, at 3 A.M. on December 23, 1936, because of increasing dyspnea, fluoroscopy was done and revealed almost complete collapse of the right lung.

RECORD OF PNEUMOTHORAX ON RIGHT SIDE

Date	Initial Pressure	Amount of Air in Cc.	Final Pressure	
		Refills		
12–17–36	−6 − 3	100	−6 − 1	
12–22–36	−11 − 3	250	−7 − 1	
		Aspirations		
12–23–36	−4 + 4	1800	−12 − 3	Spontaneous pneumothorax at 3 A.M. Also 800 cc. removed from left side.
	−4 + 2	1100	−8 − 2	
	−5 + 2	1800	−10 − 4	
12–31–36	−6 − 2	500	−10 − 2	
1–15–37	−4 − 0	600	−10 − 4	9:00 A.M.
	−12 + 2	800	−14 − 4	7:55 P.M.
1–16–37	? + 16	800	−14 − 4	8:30 A.M.
	−6 + 4	900	−12 − 4	4:55 P.M.
	−6 + 4	900	−16 − 4	7:30 P.M.
	−5 + 3	550	−14 − 12	10:55 P.M.
1–17–37	−8 + 4	900	−12 − 4	1:40 A.M.
	−8 + 3	1100	−16 − 3	8:45 A.M.
	−10 + 3	900	−12 − 4	4:00 P.M.
	−8 + 3	800	−12 − 4	8:00 P.M.
1–18–37	−8 + 2	500	−14 − 4	1:00 A.M.
	−7 + 2	900	−18 − 4	11:30 A.M.
	−6 + 5	700	−16 − 5	2:00 P.M.
	−7 + 3	700	−17 − 4	11:50 P.M.
1–19–37	−7 + 4	900	−16 − 4	9:30 A.M.

Beginning then and continuing for thirty-six hours a total of 4,700 cc. was removed from the right side and 800 cc. from the left. Five hundred cc. more were withdrawn on December 31, 1936 and the leak apparently closed. Subsequently on January 15, 1937 it reopened and between then and January 19, 11,950 cc. of air were withdrawn and a needle was left in place. Despite this she died, having been cyanotic for the preceding forty-eight hours.

Case IV developed a fistula that from its beginning was so large that it was impossible to maintain a negative intrapleural pressure.

Case IV, Mr. L. K. Tuberculous empyema, right side, with unsuccessful attempt to control by oleothorax. Readings include amount of oil given and later withdrawn.

Date	Initial Pressure	Treatment	Final Pressure
11–21–38	−4 + 2	800 cc. cloudy fluid removed − 900 cc. gomenol in olive oil instilled	−4 − 2
1–5–39	−6 − 10	Thoracentesis of 750 cc. oil, 525 cc. cloudy fluid. Air given until pressure was −8 − 3	−8 − 3
1–13–39	+3 + 8	100 cc. air removed	−8 − 0
		Rose to	−5 + 3 after few minutes
2–7–39	+5 + 9	200 cc. air removed	−7 + 1
		Changed to	− 3 + 3 when cleared throat

In this case the opening was large enough so that merely clearing the throat or waiting a few moments was enough to alter the pressure materially. In perforations larger than this the administration or withdrawal of large amounts of gas will produce no change in pressure.

In certain instances in which rupture is suspected, a highly negative intrapleural pressure may be produced intentionally to see if there is a leak. Such a method cannot differentiate between a fistula and a leak in the apparatus. Probably it is a safe test provided the pressure is not carried too low. I doubt that any harm follows the use of pressures of −10 to −12 cm. of water. Theoretically, however, a very low negative pressure might result in reopening a fistula which is sealed by fibrin, and it hardly seems justifiable to take the chance of reopening a fistula just to make a diagnosis.

Gas Analysis. The first instance which I have been able to find of the analysis of the pleural gas in a case of bronchopleural fistula is the one described by John Davy before the Royal Society of London in 1823. Because of the historic interest of this case, I am quoting at length from it. The patient also had a spontaneous tension pneumothorax and an empyema.

"Abraham Iredill, of the 7th regiment of Foot, aged 30, was admitted into the General Military Hospital at Fort Pitt, Chatham, on the 15th of January last, labouring under phthisis pulmonalis, and invalided on account of it.

"His disease exhibited some peculiarities, the cause of which was not discovered during life, the chest not having been minutely examined by exposure and percussion, owing to the severity of cold at the time and the hopeless state of the patient, evidently on the brink of the grave. He expired on the 11th of February, and his body was inspected the day following, fourteen hours after death.

"The right side of the chest exhibited a great degree of fulness, and it emitted, when struck, a hollow sound. On carefully opening the abdomen, the diaphragm was found protrading into the right hypochondrium, exhibiting a surface convex, and almost conical instead of concave; and it was tense and tympanitic. The right lobe of the liver was pressed into the epigastrium, and rested on a portion of the stomach and duodenum and a part of the transverse colon. Owing to the pressure of the liver, the pyloric portion of the stomach was removed from its natural situation to the left iliac region, where it rested on the upper part of the sigmoid flexure of the colon . . .

"The body was put into a bath, and a small opening was made, under water, with a scalpel, into that part of the right pleura, best adapted by situation to allow the escape of air. Air issued out in abundance: 212 cubic inches were collected in receivers, and about 13 cubic inches escaped, making altogether the enormous volume of 225 cubic inches (almost 3700 cc.).

"On inflating the lungs with a double bellows through an opening into the trachea, the right lung became much expanded, and air was found to pass freely from the lung into the pleura through an ulcerated opening in the upper part of the superior lobe.

"The right lung was carefully dissected out. In the upper part of its superior lobe 'a tubercular excavation,' or vomica, was found, of the capacity of about four ounce measures, which communicated with the aspera arteria by a large bronchial tube, the ulcerated end of which terminated in the side of the excavation opposite to the openings by which the vomica communicated with the pleura.

"On examining minutely the communication between the cavity of the chest and the lung, a kind of valvular structure was discovered, which would allow of air being pumped into the pleura in the act of inspiration, but not of its escape in expiration, owing to which, no doubt, the accumulation of air in question, took place . . . The cavity opened into the cavity of the chest by a hole in the pleura pulmonalis about the size of a crow-quill, and into the vomica by three smaller holes in the substance of the lung, not corresponding with the former, so that a probe could not be passed from one into the other in a straight line; and, consequently, when the surfaces of the sinus were pressed together by the compression of the air in the pleura in the act of expiration, the communication through which the air entered was closed, and its exit prevented."

The water of replacement from the right pleura when settled showed 1 ounce of white sediment.

"The air collected from the pleura had not the least foetor, nor indeed any smell. It extinguished flame, and was not inflammable. Examined by means of lime-water and phosphorus (which was sublimed in it without effect) 100 parts of it were found to consist of 8 carbonic acid gas and 92 azotic gas.

"It seemed almost demonstrated that the air was atmospheric air altered.

"The next question that presented itself was, how the alteration had taken place; what had become of the oxygene that had disappeared; whence the carbonic acid gas with which the azote was mixed?"

The observer clearly described (1) tension pneumothorax and (2) valvular fistula, and he drew the conclusion that the air was atmospheric air altered. When we consider the date, we are also impressed by the accuracy of the gas analysis. To answer his own question regarding the fate of the "oxygene" and the presence of the carbonic acid gas, he carried on a series of experiments with dogs.

Since Dr. Davy's original observation, many other workers have published studies on the composition and changes of air in a pneumothorax space under varying conditions. The reader is particularly referred to the articles by Matsuzawa.[21-24]

When air is injected into the pleural space, there is rapid diffusion through the pleura of all component gases until an equilibrium is reached. If it be atmospheric air which is injected, carbon dioxide will diffuse into the space and oxygen diffuse out. Inasmuch as carbon-dioxide diffuses much more rapidly than oxygen, the volume of a pneumothorax will be temporarily greater shortly after a refill than immediately upon its completion (Dautrebande and Spehl[9]). Carbon dioxide will also reach a stationary level sooner than oxygen, although even in the presence of marked pathology both will have practically reached an equilibrium in twenty-four hours. The rate of diffusion depends upon many factors and, also, the final levels of the gases vary according to whether the pneumothorax is "dry" or whether there is fluid, and, if fluid, whether or not it is sterile, or contains tubercle bacilli alone or tubercle bacilli plus secondary organisms.

In general, in dry pneumothorax the oxygen level is higher and the carbon-dioxide level lower than when fluid is present.

Table II

Values of Gases under Varying Conditions of Pneumothorax without Fistula

	N Per Cent	O_2 Per Cent	CO_2 Per Cent
1. Atmospheric air	79	21	.03
2. Dry pneumothorax			
Birch[5]		.9 –4.5	5.85–10.0
Coryllos, Konterwitz and Levine[7]		1.0	8.0 –10.0
Coryllos and Welkind[8]		3.8	6.0 – 9.0
Harpöth and Gad[14]		2.0 –6.0	4.0 – 8.0
Matsuzawa[21,23]		.88–3.5	5.98– 7.94
3. Fluid			
Birch[5]		.4 –4.31	9.84–14.7
Coryllos, Konterwitz and Levine[7]			14.0 or more
Coryllos and Welkind[8]		.0 – .4	10.0 –13.0
Harpöth and Gad[14]		.0 –2.0	8.0 –12.0
Matsuzawa[23]		.04–1.5	10.0 –13.0

Also, in the presence of fluid there is a further variation in that the more acute the pleurisy or empyema, the higher the carbon-dioxide level. The table below lists the values given by several different authors for oxygen and carbon-dioxide under varying conditions.

Inspection of the above table reveals that while there are differences, there is fair general agreement.

In dry pneumothorax we have a right to expect an oxygen content of 1 to 5 per cent and a carbon-dioxide content of 6 to 8 per cent. In the presence of fluid, oxygen falls to low levels, rarely being as high as 2 per cent, while carbon-dioxide ranges from 8 to 14 per cent.

With these figures in mind, let us consider the effect of a leak. In the presence of a fistula there will be a tendency for the intrapleural air to approach the composition of alveolar air: N—80 per cent; O_2—15 per cent; CO_2—5 per cent. Any leakage in the thoracic cavity lowers the

carbon-dioxide percentage but increases the oxygen percentage in the imprisoned gas (Harpöth and Gad[14]). The extent of the change will depend upon the size of the fistula and whether or not it is open constantly or intermittently.

By keeping in mind the above factors, it is not too difficult to interpret the findings on single or successive gas analyses.

In a pneumothorax without fluid, any oxygen reading above 5 volume per cent with a carbon-dioxide reading below 6 volume per cent strongly suggests perforation, if the analysis is made at least twenty-four hours following the last refill. In the presence of fluid, an oxygen reading above 2 per cent and a carbon dioxide reading below 10 per cent is of similar import. In each instance, the higher the oxygen and the lower the carbon-dioxide, the more likely it is that a perforation is present and the larger it is apt to be.

Details of technic may be found described by Matsuzawa,[23] Bettman,[4] Birch,[5] Coryllos and Welkind,[8] Harpöth and Gad.[14]

If the fistula is open at the time a sample is taken, the findings by gas analysis are apt to be clear cut. However, if the fistula is very small or if it is temporarily closed, the methods of Bettman[4] and Harpöth and Gad[14] will help. They consist in comparing values for gas content before and after a marked negative pressure is produced. In the absence of fistula the two readings for each gas will be very nearly the same. If there is a fistula through which outside air has entered during the interval, there will be a marked discrepancy in the readings. The dangers of highly negative pressures have been discussed previously.

In many cases where a single analysis is equivocal, serial analyses over a period of several days, even without the use of highly positive or negative pressure, will reveal the presence of the leak.

In this paper a great deal of time has been given to the description of gas analysis as a diagnostic aid in bronchopleural fistula. It has been shown that in many cases the diagnosis can be made in no other way. However, when it comes to treatment, any case in which the fistula has to be proved by gas analysis might as well be treated as though a fistula did not exist.

X-ray or Fluoroscopy. This type of examination is of value in fistula in revealing: (1) More collapse of the lung than might be expected from pneumothorax refills alone; (2) distortion of the mediastinum away from the collapsed side; (3) depression of the leaf of the diaphragm on the side of the fistula.

All of these findings are relative and while a fistula may be suspected from a single examination, it is difficult to prove on less than two observations, one before and one after the occurrence of the perforation. And these two observations are of maximum value only when we know whether or not the patient has had a refill in between and, if so, how much air was given and what the pressures were. Not infrequently after the administration of an initial pneumothorax or, less commonly, after a refill, *x*-ray or fluoroscopic examination will reveal a collapse of the lung out of all proportion to the amount of air given. In these cases, presumably the pleura has been wounded by the needle and a traumatic pneumothorax has developed. Fortunately, most of these are transient. If the pleura has already been thickened by an inflammatory process or if the intrapleural pressures were nearly neutral or positive, the changes observed following perforation would probably be less marked than if the perforation had occurred while the intrapleural pressures were still relatively negative and before the pleura had become thickened.

Case 1 illustrates a tension pneumothorax. The film of December 22, 1936 revealed bilateral pneumothorax without displacement of the mediastinum, whereas the fluoroscopy of 3 A.M. on December 23, 1936 revealed "almost complete collapse of the right lung."

Observations to be comparable have to be made in the same phase of respiration because, normally in pneumothorax, on

expiration there is a tendency to distort the mediastinum to the opposite side, with its return on the succeeding inspiration.

In attempts to localize the site of a fistula after thoracoplasty, we have injected an opaque medium, such as sodium iodide or iodized oil, into the empyema pocket, turned the patient on his opposite side and taken a film after a few minutes, and have found the opaque medium in the tracheobronchial tree.

Thoracoscopy. In our hands this has been valueless to determine the presence of a fistula. However, in a few instances it has been of value in determining the site of a fistula when its presence is already known. We have had no experience with the treatment of a fistula by thoracoscopy although instances of its successful use appear in the literature.[11,13]

TREATMENT

Treatment consists not in treating the fistula *per se*, but in treating the complications. In many instances, such as puncture of a lung with a needle, no treatment is indicated; also, this is true in other small or intermittent communications. Undoubtedly many of the small fistulas heal spontaneously.

The complications requiring treatment are: (1) tension pneumothorax, (2) drainage of pleural fluid into the bronchial tree, and (3) infection of the pleural space by (a) tubercle bacilli, or (b) pyogenic organisms.

Treatment may be divided into (a) immediate and (b) delayed. Immediate treatment may be needed to control any of the above complications, but once they are controlled, all treatment is concerned with the condition of the underlying lung and pleura, which are the important factors. The lung is frequently heavily infiltrated and almost all constant fistulas are associated with an underlying empyema which will heal only by obliteration of the pleural space. When a fistula is present, the chance of re-expansion is slight. Accordingly, all treatment is based upon the fact that the patient will probably come to thoracoplasty if his condition warrants, as this is the only form of treatment which adequately controls the pulmonary disease, the empyema and the fistula.

Tension Pneumothorax. This may be described as a condition in which, during ordinary respiration, intrapleural pressures are at all times positive. This is frequently due to a ball-valve fistula which will allow air to pass into the pleura but not out. If a tension pneumothorax develops, the patient and surgeon may be presented with an emergency which, if not relieved, causes death. There is usually time for an *x*-ray or fluoroscopic examination, although Barnwell[3] records an instance of the patient dying during the trip from the fluoroscopy room. He strongly warns against taking time for *x*-ray in critical cases. The diagnosis is best made and combined with treatment by taking the intrapleural pressures and withdrawing air until the dyspnea is relieved or until the intrapleural pressure on inspiration is down to minus 10 cc. of water. Sometimes when a physician sees such a patient, he is in extremis. Air should be withdrawn, without delay, by any needle and syringe even if there is no manometer handy. Failure to aspirate promptly has cost many a life. Once the dyspnea is relieved, there is time to plan further treatment. It should be mentioned that frequently the pressures build up quickly again, requiring repeated withdrawals of air. In such an instance, a fairly effective safety valve may be made by taking a No. 18 gauge needle and grinding down the point until blunt. This is then passed through a thin cork. The needle is then inserted into the pneumothorax pocket near its upper point and the cork strapped to the chest. The needle is adjusted so that the point is barely within the pleura. One of two things may be done: (1) A finger from an old surgical glove may be tied over the end of the needle and the end of the finger opened; or (2) a tube may be connected to the needle and led to a sterile flask containing a mild antiseptic.

This flask should be a foot or two below the level of the patient and the end of the tube should be 1 to 2 cm. below the water level.

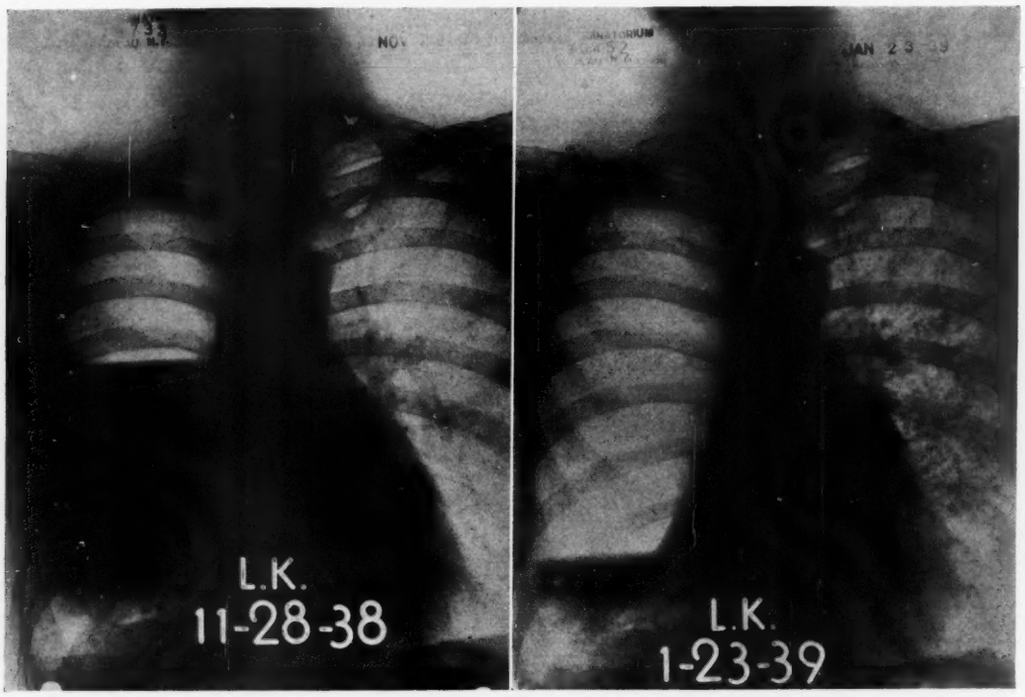

Fig 5. Fig. 6.

Figs. 5 and 6. Case IV. Tuberculous empyema, right. Figure 6 shows the spread of disease in the opposite lung which followed the development of a bronchopleural fistula on the right, with expectoration of fluid.

Either method will prevent the building up of a high positive pressure as long as the needle is not blocked by clotted blood or serum. Care has to be exercised to see that the needle is not blocked and that it does not become loosened and displaced out of the pleural cavity.

Drainage of Pleural Fluid into the Bronchial Tree. Wherever there is purulent pleural fluid draining through a bronchus, there is always the possibility of seeding tubercle bacilli to the same or opposite lung. In addition, even in those cases in which spread does not take place, there is the constant annoyance with disruption of sleep by the continued coughing up of small amounts of pleural fluid. These perforations are usually in the upper portion of the lung and manifest their presence only when the patient lies down and the opening comes below the fluid level. Immediate aspiration should be done and all fluid removed. Within the next few days, and before fluid has had time to reform, drainage should be performed. It is simple and usually adequate. It is not safe to depend on repeated aspiration to keep the pleural cavity empty. Case IV illustrates this point.

Case IV. Mr. L. K., a man of 50, had a pneumothorax on the right induced August 11, 1937. Following two pneumonolyses he developed fluid which became positive for tubercle bacilli eight months later. With the appearance of the fluid the cavity could not be seen and the sputum became temporarily negative. To control the tuberculous empyema, oleothorax was begun in November 1938, and by the twenty-eighth of the month (Fig. 5) fluid and oil had risen to the third rib and fluid formation had apparently ceased. On January 2, 1939, the patient suddenly coughed up a large amount of fluid which contained gomenol. All pus and oil were aspirated *and on January 5 there was no fluid remaining in the cavity*. However, fluid did reform and on January 23, 1939, there was a spread to the left lung. (Fig. 6.) With repeated aspirations the patient developed a pleurocutaneous fistula. Intercostal drainage was

finally performed. Later, rib resection was necessary. He has never since been in shape for thoracoplasty.

ated. When there is no fistula, there is a choice of re-expansion of the lung or of thoracoplasty, depending upon the condi-

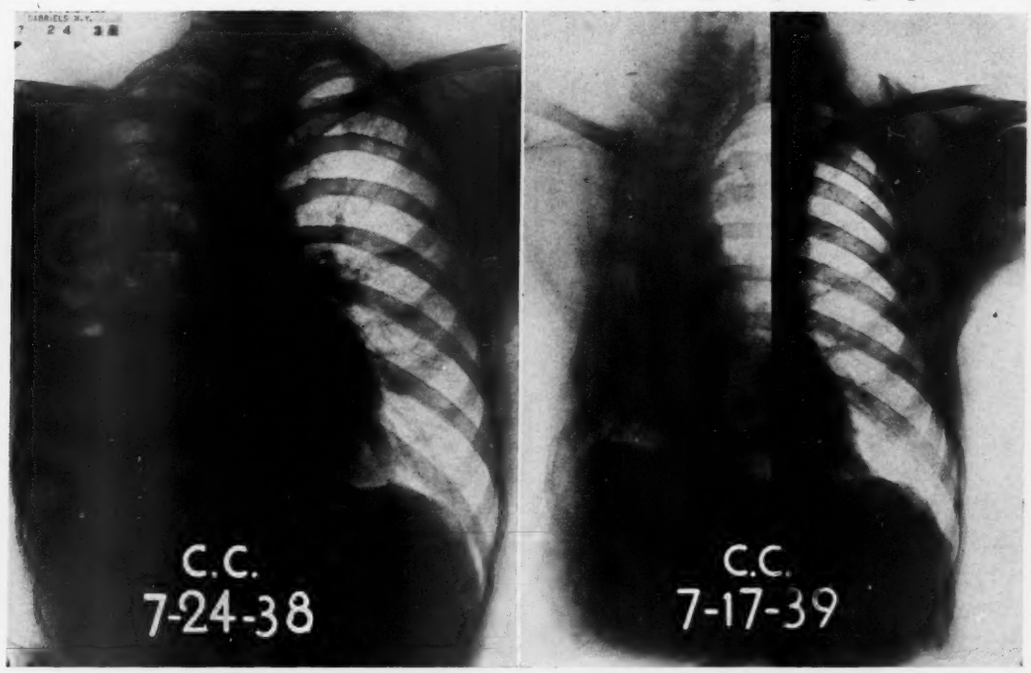

Fig. 7. Fig. 8.

Figs. 7 and 8. Case III. Figure 7, tuberculous empyema, right, three days following positive diagnosis of perforation but probably more than two weeks after the occurrence of the initial leak. First-stage thoracoplasty was performed the following day. Figure 8, one year later.

It would seem that if he had had intercostal drainage when the perforation first occurred, he might never have had the spread to his left lung. Had his perforation been smaller, there would have been less likelihood of his disease spreading.

Infection of the Pleural Space. It has been shown earlier in this paper that in almost every instance of fistula there is a tuberculous infection of the pleura but secondary organisms cannot always be demonstrated. Although tuberculous infections can be very virulent, usually they are less severe than the mixed infection empyemas and their mortality rate is approximately one-half of the latter. We shall discuss separately the treatment of (a) tuberculous empyema and (b) mixed infection empyema. The discussion will be limited to general principles.

(*a*) *Tuberculous Empyema.* The author has previously outlined his theories on the treatment of tuberculous empyema.[30] In general, the pleural space must be obliteration of the lung. In the presence of a fistula the possibility of re-expansion is very slight and thoracoplasty should be done as soon as possible. In the majority of instances, drainage is contraindicated unless the fistula is large enough to allow flooding of the bronchial tree, or unless the tuberculous process is extremely virulent. In such instances it should be treated as a mixed infection. Early obliteration of the space will frequently forestall mixed infection empyema with its long, trying period of toxemia, and in many instances it will be possible to proceed with thoracoplasty as soon as the fistula manifests itself. When possible, this is ideal treatment. In Case III, previously mentioned, this was possible.

Mr. C. C., age 27, was a patient of Dr. John Hayes, at the Sanatorium Gabriels. Pneumothorax on the right was begun February 10, 1938. It was not entirely successful and a phrenic was added on March 26, 1938. The pneumothorax was still ineffective and accordingly was discontinued on April 29. During this

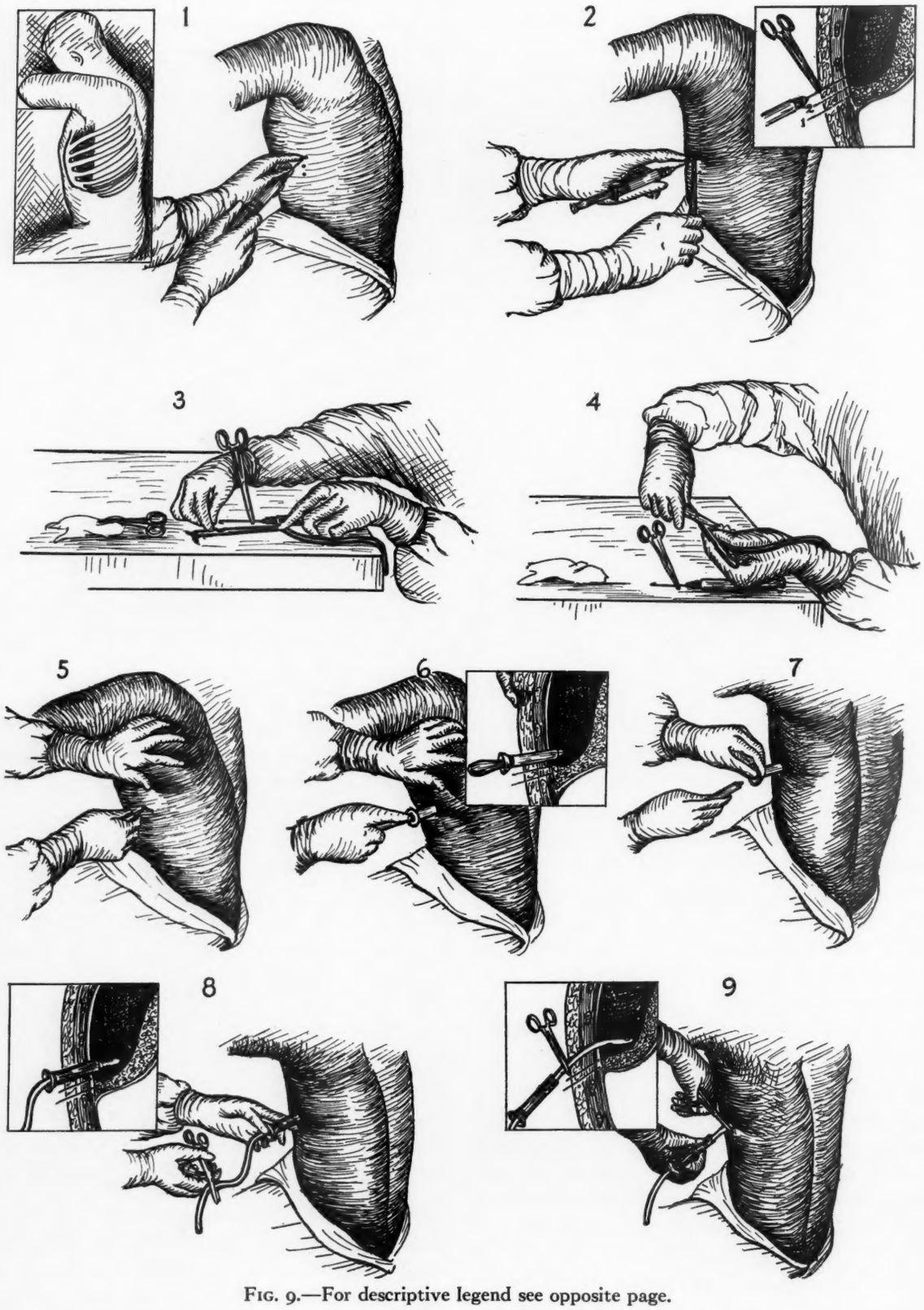

Fig. 9.—For descriptive legend see opposite page.

short pneumothorax, fluid had developed and tubercle bacilli were found. It became frank pus by June, 1938, and re-expansion was attempted

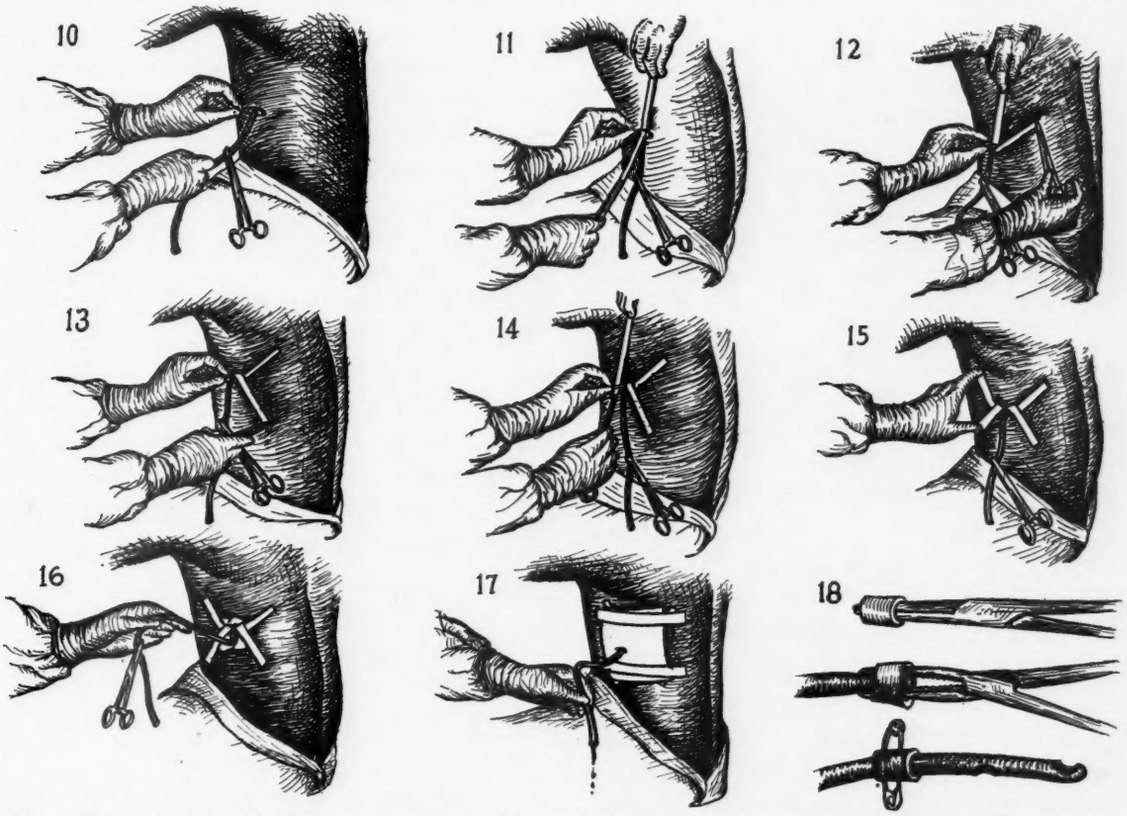

FIG. 9. Technic of introducing intercostal tube. The position of the patient is shown in 1, as well as the site of aspiration. We aspirate from below upward until the pocket is tapped at its lowest point. In 1 and 2 it is seen that there are two dry taps before the pocket is entered. When the pocket is entered, the thickness of the thoracic wall is measured on the aspirating needle by clamping it with the hemostat (2). Several additional eyes are cut in the catheter. From the proximal eye a distance is measured equal to the thickness of the chest wall (3). This point is marked by nicking the wall of the catheter, care being taken not to go into the lumen (4). After the catheter is marked, the trocar is inserted. The skin is held taut with the left hand and the point selected is nicked with the scalpel (5). The trocar and cannula are then introduced (6). The trocar is removed, the gush of pus being controlled by the thumb (7). The previously prepared catheter is inserted for a further distance than is necessary (8). The cannula is withdrawn, the tube being held in place by a hemostat, and the catheter is then withdrawn until the mark previously made in 4 is now at the skin margin (9). A safety pin is passed through the catheter at the site of marking and is fastened to the skin by adhesive straps as described by John Alexander (10–17). It keeps the field cleaner if the tube is kept clamped throughout this stage.

Placing a safety pin directly through a catheter is a disadvantage wherever closed system drainage is necessary, as there is always leakage. Accordingly, when catheters have to be changed, it is simple to insert a catheter with a collar on it. The safety pin is then put through the collar. The method of slipping on the collar is illustrated in 18. A bit of tubing, the same size as the catheter, is taken. The end of the catheter is grasped by a hemostat and the catheter stretched. The collar will then slide easily to any point. It will not slip and the tube will not leak.

preliminary to thoracoplasty. During the re-expansion the patient had pleural lavage. During July a highly negative pressure was obtained, air and fluid being withdrawn to maintain it. The first suggestion of a possible leak occurred on July 12 when the initial pressure was plus 6, plus 8. It was brought to minus 24, minus 18 by the aspiration of 450 cc. of air. However, three days later the pressure was again positive. Positive diagnosis was not made until July 21 when during the irrigation of the pleural cavity the patient started to cough and raised sputum colored with gentian violet. The following day his temperature reached 102°F. Plans were made for immediate thoracoplasty and this was carried out on July 25. Immediately preceding collapse, the pleural cavity was emptied by aspiration anteriorly. (This is quite important because, unless the

pleural cavity is emptied immediately before the patient goes on the table, it is possible to have a spill of a large quantity of infected fluid into the opposite lung.) His convalescence was uneventful. He is now well and working. Figure 7 was taken three days following the positive diagnosis of perforation but probably more than two weeks after the occurrence of the initial leak. Figure 8 shows the patient's condition on July 17, 1939, ten months following completion of his thoracoplasty with obliteration of the pleural space and no evidence of the presence of fistula.

(*b*) *Mixed Infection Empyema.* The treatment here consists of adequate dependent drainage, supportive treatment and waiting until the patient is in shape for thoracoplasty. In the presence of a fistula, irrigation with various antiseptics has to be used with the greatest caution and is of limited value.

The technic of drainage merits discussion. It is well recognized that in the treatment of empyema the drainage tube should be put into the dependent portion of the chest. However, if it is put in posteriorly, it is uncomfortable for the patient to lie upon. In tuberculous empyema with fistula there is another factor in choosing the site for drainage. That is that thoracoplasty will probably be necessary. Accordingly, it is advantageous to keep the site of drainage away from the site of the thoracoplasty incision so that contamination may be postponed as long as possible. Therefore, the site of election for drainage is slightly in front of the midaxillary line at the lowest possible interspace at which pus can be aspirated. When the drain is introduced in this position, it is possible to empty the pleural space completely by having the patient sit up and lean to the side being drained. With a drain in this position, it is not difficult to keep the thoracoplasty wound uncontaminated through the upper stages.

Drawings (Fig. 9) made from photographs taken during the insertion of a tube in Case 11 illustrate the author's method of procedure in all cases of tuberculous empyema requiring drainage. In many instances this is adequate. In other cases there will be much pain from pressure on the intercostal nerve above. To give relief it is necessary to block the nerve. The author prefers to combine this with the resection of about one and one-half inches of rib at the lowest portion of the pocket in the midaxillary line. Portions of the same intercostal nerve and the one above are taken and the wound closed around the tube. Over a period of time the wound will heal tightly around the tube. When there is no expectation of further treatment and when the patient will continue to drain indefinitely, it is possible to make a larger opening and invert the skin edges. Once such a wound is healed, no tube is needed and the patient can get around with the use of a pad.

Grateful acknowledgment is made to Mrs. Madge Vorwald for the illustrations of the technic of intercostal drainage and to Mr. Frank Bucci for taking the photographs which formed the basis for the illustrations.

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BENIGN LESIONS AT THE LOWER END OF THE ESOPHAGUS

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CARDIOSPASM

ACHALASIA and cardiospasm are two of the most commonly used terms referring to the dilatation and hypertrophy of the esophagus and spasm of the musculature of the cardia. Passage of food normally from the esophagus to the stomach is partially or completely blocked; yet in most instances no true mechanical obstruction can be demonstrated. A few of the many other terms used are phrenospasm, mega-esophagus, preventriculosis, hiatal esophagismus, simple ectasia of the esophagus and esophagectasia.

Willis described the syndrome in 1674, followed by Hoffmann in 1733. Purton probably reported the first case in 1821 and since then the multiplicity of terms to describe the disease, of hypotheses concerning its cause and of methods to effect its cure well indicate the highly controversial nature of the syndrome.

Etiology. The pathogenesis of the disease is unknown. Many hypotheses have been advanced, but the one suggesting autonomic imbalance seems most acceptable. Cardiospasm in cats can be produced, according to Knight, and Knight and Adamson, by bilateral section of the vagus nerves. The development of the obstruction can be forestalled by immediate removal of the sympathetic fibers and, if obstruction already has occurred, relief may be obtained by sympathectomy. Sympathectomy on human beings, however, as reported by Ochsner and DeBakey, and Skinner and one of us (H. K. G.), has not been satisfactory. Rake demonstrated degenerative changes in the ganglion cells of Auerbach's plexus at the lower end of the esophagus, suggesting an alteration in the neuromuscular control of the sphincter. Ochsner and DeBakey have suggested a chronic deficiency of vitamin B as the cause of this degeneration. Some authorities have based their impression of the causation of cardiospasm on the presence of a cardiac sphincter; yet others have demonstrated as convincingly its absence. Sauerbruch and Haecker, in 1906, suggested that the crura of the diaphragm acted as a vice, constricting the esophagus.

Pathology. There are definite pathologic changes associated with the disease. The esophagus actually may be lengthened, but it is characteristically dilated and its walls are thickened. Lambert described three typical shapes of the esophagus: (1) fusiform, (2) flask-shaped and (3) sigmoid-shaped. Some cases of ulcerative esophagitis at the diaphragmatic level have been noted.

Symptomatology. The characteristic symptoms of the disease are dysphagia, regurgitation and epigastric pain. At least at its inception the majority of the patients are emotionally unstable, with many complaints other than those referable to cardiospasm. Plummer described three stages in its clinical course: (1) cardiospasm without regurgitation of food, (2) cardiospasm with immediate regurgitation of food and (3) cardiospasm with a dilated esophagus and retention of food in the dilated part. It apparently makes little difference if the ingested food be solid or liquid. In the early stages regurgitation is prompt and partial and there is no marked loss of weight. As the tone of the esophagus decreases and dilatation ensues, regurgitation may be delayed for hours and the emesis may be of retention character. The pain is usually

substernal, usually occurs immediately on ingestion and swallowing of food and usually is referred upward to the throat. Dysphagia and pain may occur long before the onset of regurgitation.

Therapy. The treatment of cardiospasm has been reviewed in recent articles by Skinner and one of use (H. K. G.) and Ochsner and DeBakey, among others. The former authors mentioned 1,200 cases of cardiospasm encountered at the Mayo Clinic, with surgical treatment necessary in but seven instances. Moersch, in 1933, reported 810 cases from the same clinic, 805 patients having been treated conservatively by dilatation from above. Seventy-one per cent of the 670 patients traced were well. If the dilatation of the esophagus is sigmoid-shaped as noted by Lambert, surgical treatment may be necessary in order to obtain relief.

Ochsner and DeBakey divided the various types of radical surgical operations into four groups:

1. Operations directed at the dilated esophagus: (a) Excision of the wall, (b) esophagoplication and (c) esophagostomia thoracica. These procedures usually proved to be unsatisfactory.

2. Operations directed at the cardia: Such operations seem more rational. In the cases collected by Ochsner and DeBakey, the results from retrograde and transgastric dilatation are no better than those reported for more conservative treatment. Cardiomyotomy and cardioplasty are two of various plastic procedures on the cardia, and in the collected cases, gave favorable results. Esophagogastrostomy seems the most rational and in the hands of experienced operators gives good results. The transperitoneal route has been found to be preferable to the transpleural route and in most instances should be used. More than 90 per cent of the patients in the collected cases report good results from esophagogastrostomy.

3. Operations directed at the diaphragm: Phrenicotomy and phrenico-exeresis have been done, with uniformly poor results.

4. Operations directed at the nerve supply: These operations were suggested by the assumption of some derangement in the autonomic balance. Vagotomy and sympathectomy seem to be most popular. The results have not been as encouraging as those from esophagogastrostomy. In the collected cases of Ochsner and DeBakey, seven recurrences, three deaths and only one satisfactory result were noted.

EPIPHRENAL DIVERTICULA

Diverticula of the esophagus in various portions of its length are fairly common. They are best classified by their anatomic position and may be divided broadly into three groups: (1) the pharyngo-esophageal, (2) the epibronchial and (3) the epiphrenal or supradiaphragmatic. These may be either true diverticula or false, depending on whether there is protrusion of all coats of the esophagus or merely herniation of the mucous membrane with its submucous coat. Another well known method of classification is that of dividing the diverticula into two types: (1) the traction type, represented by the epibronchial group and occurring at the level of the bifurcation of the trachea; (2) the pulsion type, represented by both the esophagopharyngeal group, located at the hypopharynx, and the epiphrenal group, located at the cardia.

The pulsion supradiaphragmatic diverticula occur in the lower third of the esophagus, usually because of a congenitally weakened esophageal muscular wall. Many authors (Raven) have expressed the opinion that cardiospasm is frequently associated with these diverticula. They occur much less frequently than either of the other types, Granet collecting only forty-nine cases in 1933, and Vinson reporting twenty-four more from the Mayo Clinic in 1934. Scattered reports of epiphrenal diverticula have appeared since, usually included in the larger group of esophageal diverticula, most of which were epibronchial in character.

Ludlow reported "preternatural pockets" in the esophagus in 1764, but the first

reference to epiphrenal diverticula was made by DeGuise in 1833, and roentgenographic diagnosis is mainly responsible for the discovery of most true cases of supradiaphragmatic diverticula.

Etiology. The causation of these diverticula is not known. They are of the pulsion type and the location of the weakness and character of the opening into the sac led Carman to suppose that the majority were congenital. Along with this congenital weakness in the esophageal coat, two further factors may aid in the enlargement of the sacs, once they are formed: (1) the positive intra-esophageal pressure during swallowing and (2) the negative intrathoracic pressure. They usually present on the right side, and this is explained by the anatomy involved (Dessecker). Since the esophagus curves to the left in its lower third to reach the stomach, the impetus of food boluses may be borne by the outer, lower right side of the curve. Once started, the diverticula usually enlarge in a downward direction, toward the right.

Symptomatology. The symptoms are vague and insidious. A feeling of epigastric pressure may precede more pronounced symptoms by months. The cough attending epibronchial diverticula is usually lacking. The actual symptoms depend on the gradual increase in size of the sac. They may be rendered vague by co-existent achalasia. When a large diverticulum is encountered, substernal pain, dysphagia and vomiting of retention are marked. Since the neck of the sac is narrow, the usual complaint is that of vomiting decomposed food, often ingested days before. Often such regurgitation interferes with sleep and is rather characteristic of these sacs. Such putrefaction and stasis cause inflammation of the sac with increased severity of symptoms. Emaciation is usually marked in well advanced cases. Large diverticula may occlude the esophagus by pressure. The diagnosis is rendered usually by roentgenologic examination.

Treatment. The location of the sac usually makes conservative treatment imperative. Often treatment by occasional lavage from above in mild cases is sufficient. Dilation of the esophagus with bougies is attended with success, especially if there is attendant cardiospasm. Extirpation of the sac by surgical means is difficult and imposes considerable risk because of the great probability of soiling the mediastinum or the pleural space or both. In 1933, Lahey first reported a conservative plan of treatment surgically in those cases in which the size of the sac demanded such intervention. He suggested fixing the dome of the freed sac high in the pleural gutter so that it parallels the esophagus with its neck and opening pointing downward. The hazard of pleural contamination and mediastinitis is averted, and food passes readily by the opening unless the patient is in the Trendelenburg position. In four such cases the patients were treated in this manner at the Lahey Clinic. Turner[49] has suggested inverting the diverticulum into the lumen of the esophagus and suturing it in this position. The avoidance of pleural and mediastinal soiling has been accomplished by the method described by Lahey. Subsequent examination is stated to have shown that the sac of the diverticulum that protruded into the esophagus in most instances had undergone atrophy after a short time and was represented by a small structure that was of no anatomic or clinical significance.

ESOPHAGEAL VARICES

Esophageal varices depend for their development both on the obstruction of the portal and lienal veins and on the poor support rendered by the loose connective tissue of the submucosa of the esophagus. The inherent dangers of such varices lie in their tendency to bleed and their inaccessibility for treatment. McIndoe, Preble, Rivers and Wilbur have emphasized the frequency with which fatal hemorrhage attends these varices.

Etiology. The causation of the lesion is not clear, for the conditions most often associated with it, that is, Banti's disease and cirrhosis of the liver, do not present

clear-cut clinical entities. Kegaries and McIndoe have well demonstrated both the anatomy and the physiology of the region. When the portal system has become obstructed, an effort is made to establish collateral circulation and the esophageal veins, because of the connection with the coronary vein and vasa brevia of the cardia, dilate and protrude into the lumen of the esophagus. Varices may be shown to be present in conditions other than the two already mentioned; yet the threat of hemorrhage has been found to be less apparent. Varices of the esophagus may be found in patients who have goiter, following trauma or severe esophagitis. In splenic anemia it is thought that the explanation of the development of varicose veins of the esophagus lies in the connection of the spleen and cardiac portion of the stomach by the vasa brevia.

Diagnosis of the lesion was made much simpler and more accurate by the report of Wolf, in 1928, describing a method of demonstrating the varices roentgenologically. The technic has been steadily improved and perfected as described by Schatzki. Kirklin and Moersch, in 1931, called attention to the value of combining esophagoscopic and roentgenologic examinations to determine the extent of the lesion.

Treatment. Treatment of the varices may be either conservative or surgical. Conservative measures imply protection of the varices from trauma by bland, nonirritating diet, while one attacks the underlying cause. Minimal exercise to avoid strain and sudden increase in blood pressure and avoidance of the use of the stomach catheter are important. Drenckhahn suggested venesection to decrease the viscosity of the blood.

Surgical measures may be directed (1) toward removing or sclerosing the varix, (2) toward reducing as far as possible the amount of blood entering the portal system, (3) toward establishment of new and less dangerous collateral circulation and (4) toward ligation of those routes already established and contributing to the varices.

The direct removal of the varices is dangerous and unsuccessful. Moersch and others have attempted injection of a sclerosing substance into the varix but this method has not been performed in a sufficient number of cases to warrant definite conclusions being drawn. Moersch is inclined to believe that this method should be performed in conjunction with one of the other methods, preferably splenectomy.

Splenectomy has been performed for Banti's disease for years. It diminishes the total blood volume entering the portal system by about 25 per cent (Drenckhahn) and changes the route of the major portion of the blood that formerly passed through the vasa brevia. Recurrent hemorrhages from splenectomy alone are reported at 50 per cent by Pemberton. Walters, Moersch and McKinnon have reported eighty cases of splenic anemia in which treatment was by splenectomy alone, in all of which bleeding was present preoperatively. In thirty-three of these there was recurrent hemorrhage.

The establishment of collateral circulation may be attempted by omentopexy. The denuded peritoneal surface after splenectomy may offer similar relief. Such methods have not been uniformly successful and have been combined usually with ligation of the coronary vein or even splenectomy.

Ligation of the coronary vein and of the vasa brevia of the cardia in conjunction with splenectomy is a method of interrupting the collateral routes contributing to the varices. Few cases of ligation of the coronary vein alone have been reported and its value as yet is undetermined. Splenectomy combined with such ligation or with omentopexy does not prevent recurrence of bleeding from the varices in more than 38 per cent of the cases (Walters and others).

From a review of the literature it would seem that the most promising treatment,

the value of which is still undetermined, is the injection of the varices by means of the esophagoscope, in conjunction with an attempt to bring the underlying cause under control. The combination of such esophagoscopic treatment with splenectomy seems to offer the best chance of avoiding fatal hemorrhage.

ATRESIA

Congenital atresia of the esophagus occurs rarely, and then often in association with some other anomaly of the gastrointestinal tract incompatible with life. Lanman reported thirty-two cases from the Children's Hospital in Boston in 1940, and stated that he believed the total number of reported cases would not exceed 300. Rosenthal described the embryology fully and Strong and Cummins, Vogt and others have classified the various types of atresia encountered.

The classification suggested by Vogt is excellent if one is to understand and interpret the symptoms and direct attempts at surgical repair. He suggested three types: In type 1 there is complete absence of the esophagus. In type 2 both upper and lower segments end blindly and without tracheal communication. Type 3 he divided into three varieties: type 3 *a*, in which the lower segment is blind, the upper communicating with the trachea; type 3 *b*, in which the upper segment is blind and the lower communicates with the trachea; and type 3 *c*, in which tracheal fistulas are caused by both upper and lower segments. All these varieties are rare, but by far the most common is type 3 *b*.

Symptomatology. The symptoms are characteristic. Attacks of choking and cyanosis are present from birth and are much aggravated by offering milk or water. Depending on the type of anomaly present, regurgitation or choking and coughing predominate. Diagnosis is accomplished by the history and made certain by roentgenographic procedure. Contrast media should not be used because of the danger of aspiration. A small, soft nasal catheter may be introduced to determine the extent of the upper segment. A small leaden tip may be helpful. Again the type of anomaly will be suggested by the presence or absence of gas in the stomach.

Treatment. The treatment of the condition is necessarily surgical, and the time-honored method is by gastrostomy. This method is only palliative and the patient will not survive if the usually existent fistula is not repaired. As yet there is no uniformly successful method of approach to repair the tracheo-esophageal opening. Richter suggested direct attack on the posterior mediastinum twenty-eight years ago. Extrapleural and transpleural approaches are time consuming and the mortality as yet is very high. The treatment of the upper and lower segments is a controversial subject. Direct anastomosis of the two segments is the ideal method. Lanman expressed the belief that the fistula should first be closed if direct anastomosis is impossible. This is then followed by exteriorization of the upper segment and anterior gastrostomy to prevent aspiration of the overflow secretions and allow feeding. Further construction of an artificial external connection between the esophageal stoma and the gastric stoma can be delayed. Undoubtedly, improvement in technic of approach and exposure, whether transpleural or extrapleural, will brighten the outlook for future success.

BENIGN ULCERS OF THE ESOPHAGUS

Benign discrete ulcers of the esophagus are more common than ordinarily believed. They have a predilection for the lower third of the esophagus and may often be confused with gastric disease. Rivers found that 45 per cent of his patients who had esophageal ulcer gave a history of peptic ulcer. Jackson, by esophagoscopic examination, found in 4,000 cases only twenty-one peptic ulcers, but noted scars of sixty-seven others.

Although Butt and Vinson expressed the opinion that it is merely a phase of acute ulcerative esophagitis, benign ulcer of the

esophagus, exclusive of specific infection, may be classified as traumatic or inflammatory. The traumatic ulcers may be caused by foreign bodies, instrumentation, decubitus, or burns (corrosive). The inflammatory ulcers may be associated with peptic ulcer or with infectious disease.

The ulceration that follows lodgment and pressure by foreign bodies, instrumentation or the swallowing of caustic and corrosive substances can be understood. The cause of simple ulcer with no such traumatic factor is unknown. The inflammatory process surrounding esophageal ulcer is similar to that attending the gastric or duodenal ulcer so often also present.[7] The hypotheses advanced for the causation of acute esophagitis are applicable here. Regurgitation of acid contents of the stomach, heterotopic gastric mucosa, and infection from the buccal cavity are undoubted factors. Often an ulcer attends congenital shortening of the esophagus; often it is a sequel to acute or chronic esophagitis; often it precedes stricture and attends congenital stenosis and hiatal hernia.

Symptomatology. Dysphagia and pain are usually early symptoms, not to be distinguished from those of esophagitis. Pain is usually substernal, made worse by swallowing and referred directly through to the back. Cardiospasm may be present almost with sphincteric action. Esophagitis usually is associated, and may be part of the disease, as suggested by Butt and Vinson. Pain referred to the back may suggest a coincident hiatal hernia or congenitally short esophagus with thoracic stomach. Regurgitation is common but bleeding is less so. The distress may be relieved by alkalis or aggravated by the prone position. Odynophagia is common, and dysphagia may become worse if cicatricial stenosis occurs. Diagnosis is usually made, or confirmed, by esophagoscopic examination.

Treatment. The treatment cannot be specific. The general condition of the patient must be improved and the sources of infection in the mouth and throat removed. A trial of medical management similar to that for peptic ulcer must be instituted. Application directly through the esophagoscope of 10 per cent solution of silver nitrate or chlorlyptus oil has been recommended by Patterson.[33] Often the cardiospasm and stenosis will be benefited by dilatation from above by bougies.

Occasionally, severe and recalcitrant ulceration will require gastrostomy to put the esophagus completely at rest and allow adequate intake of vitamins and calories. Many of these patients approach avitaminotic states because of their prolonged and enforced partial fasting. Adequate quantities of all vitamins should be ensured.

STRICTURE

Stenosis of the esophagus may occur either because of lesions in the esophageal wall or because of extrinsic pressure. This latter type usually occurs in the lower third and has been referred to as compression stenosis. Cardiac enlargement, hiatal hernia, lymphadenopathy and aneurysm are a few factors in the production of the condition. It may be the first sign of the presence of the underlying disease. Dysphagia is the primary and distressing symptom. Diagnosis is usually difficult, but aid can be obtained by roentgenologic examination of the chest and even by esophagoscopic examination. Treatment is that of the underlying lesion, with gastrostomy if necessary to combat starvation.

Etiology. There are many causes of the esophageal stricture due to inflammation or trauma of the esophageal wall. Turner[50] included a description of a specimen of esophageal stricture quoted from Bell's "Surgical Observations" of 1816–1818. Barclay demonstrated that stricture may result from any inflammatory reaction in the esophageal wall. Caustic substances are well known agents; yet both Turner and Vinson[51,56] stated that discrete foreign bodies rarely cause stricture, even if ulceration has occurred. Peptic ulcer of the esophagus, in healing, may exert a cicatricial influence; yet Vinson[58] expressed the

belief that such ulcers cause very little stenosis. Vinson[51] also described nine cases in which esophageal stricture followed the vomiting of pregnancy. Strictures have been reported following the infections of childhood, typhoid fever, diphtheria, severe pneumonia, syphilis and tuberculous ulceration of the esophagus. Congenital shortening of the esophagus, with thoracic stomach, is frequently a cause of ulceration and stricture at the juncture of the esophagus and stomach. Such stenosis is less common than cardiospasm and carcinoma.

There remains a rather large group of cases of esophageal stricture for which no cause can be found. This is known as benign, or idiopathic, stricture of the esophagus. Vinson[53] reported 186 cases of stenosis of the esophagus in 1923, eighteen (9.7 per cent) of which had no known cause. Again in 1931 Vinson[56] reported 243 cases of stenosis of the esophagus and remarked on the increase in the number for which no cause could be demonstrated. Forty of these 243 cases (16.5 per cent) fell in this group. In more than half of the cases this type of stricture occurs in the lower third of the esophagus.

Symptomatology. The symptoms depend on the degree of stenosis encountered. Such symptoms are usually esophageal in origin and character, and the most constant are dysphagia, regurgitation and usually some substernal pain. The history of ingestion of corrosive substances is enlightening. The presence of an inlying nasal catheter or history of intubation is suggestive (Butt and Vinson). Vinson[56] on several occasions has stated that the symptoms of peptic ulcer of the esophagus usually are absent in those cases of benign stricture of unknown cause. The only significant symptom in this group of cases is dysphagia. Emaciation of starvation and avitaminotic states may occur in cases in which treatment has been neglected for a long time.

The diagnosis of the stricture, even if the cause is unknown, may be made certain by roentgenologic studies and esophagoscopy.

Treatment. The treatment usually begins with the conservative methods of dilation and bouginage. Such dilation should always be accomplished with a swallowed thread as a guide. Vinson[58] recently described the procedure minutely and urged that roentgenologic examination of the stomach itself, to detect pyloric stricture, be performed after the swallowing of corrosives. The treatment must be continued over a long period to prevent reformation of the cicatrix. Such therapy is uniformly successful for cases of benign stricture of unknown cause. Complete obliteration of the esophagus from swallowing corrosives is possible and may necessitate gastrostomy to combat starvation and allow retrograde bouginage. Prolonged attempts to pass a thread from above for guidance are urged. Vinson[58] stated that gastrostomy for such pronounced strictures carries a mortality rate of 10 per cent. Turner[50] advised that the opening for the retrograde dilation be made very near the cardia, but he expressed the belief that in the great majority of cases such conditions may be cured by painstaking and patient dilation. He has lately been urging "self-bouginage" over a period of years, for as stated previously, these strictures are always likely to recur.

There are a few cases recorded in which all such conservative methods have failed. After preliminary gastrostomy, plastic operations of the well known Heineke-Mikulicz type have been attempted with varying success. Esophagogastrostomy may be attempted if the lesion is near enough to the cardia. The strictured portion of the esophagus cannot be removed with any degree of success.

One further procedure, anterothoracic esophagoplasty, has been tried in very few recorded cases. Ochsner and Owens reviewed the literature in 1934. Eggers, Garlock and others have been attempting plastic procedures with extrathoracic management of the proximal portion of the esophagus and gastric fistula. The field is very limited in which such procedures may be used; in selected cases (1) of tracheoesophageal fistula, (2) of benign stricture of

the esophagus impermeable to all known methods of dilation, and (3) of malignant stricture of the esophagus in which carcinoma has been removed successfully.

ESOPHAGITIS

The lower third of the esophagus is exposed to infection both by organisms from the mouth, throat and nasopharynx and by regurgitation of gastric contents. Acute esophagitis is rather common as transient manifestation of the damage done by ingested caustics, alcohol or any trauma. Its occurrence as an agonal feature is shown by Bartels' study of eighty-two cases in 6,000 postmortem specimens. Butt and Vinson found 213 cases of esophagitis in 3,032 necropsies.

Detailed descriptions of the various kinds of esophagitis are easily available. Acute and chronic catarrhal esophagitis; follicular, diphtheritic, phlegmonous esophagitis; and esophagitis due to specific organisms of tuberculosis, syphilis and fungal infections, all are seen but rarely. More common is acute ulcerative esophagitis, the cause of which is uncertain.

Etiology. Butt and Vinson expressed the belief that the trauma of intubation, of vomiting or of the two combined is important. In 46 per cent of their cases an inlying nasal catheter had been associated with vomiting. Bloch reported an even higher percentage; eleven of seventeen patients who had acute esophagitis had been intubated. The frequency of the association of peptic ulcer and acute esophagitis is well known. Penner and Bernheim expressed the belief that most acute esophagitis is an agonal episode, produced by shock. The poor circulation and weak submucous and muscular structure of the esophagus favor embolism and thrombosis and these may play a part in the causation of the condition. The rôle of the nervous system in perforating lesions of the intestinal tract has been emphasized by Cushing. Pneumonia and other serious infectious diseases may have a part. Chronic esophagitis is usually only part of a syndrome of gastric disease found among persons suffering from chronic alcoholism and among those addicted to the eating of highly seasoned or very hot foods.

Symptomatology. In the series reported by Butt and Vinson 76.5 per cent of the cases were of the acute type, 14.5 per cent were rather subacute, and only 6.5 per cent were of the chronic variety. The symptoms of acute esophagitis may suggest a gastric disturbance. Substernal pain with extension to the throat during and immediately after deglutition, dysphagia and occasional hematemesis are characteristic. Diagnosis in the early stages cannot be proved by roentgenologic examination as a rule, but esophagoscopic examination can be used.

Treatment. The treatment of esophagitis is symptomatic and supportive. In view of the findings of the foregoing authors, the passage of a nasal stomach catheter in the presence of esophagitis would be foolhardy. Sedation, bland foods and parenteral fluids if necessary are helpful. Occasionally, gastrostomy to put the esophagus strictly at rest is necessary.

BENIGN TUMORS

Although benign tumors of the esophagus occur rarely, they must be carefully distinguished from the more common, and usually fatal, malignant varieties. Patterson,[32] in 1932, could collect only sixty-one cases from the literature. Vinson[52,54,55] on several occasions, and Harrington and Moersch more recently, have reported benign tumors of the esophagus seen at the Mayo Clinic.

In 7,459 necropsies these latter authors reported forty-four benign lesions. A great variety of such lesions are reported in the literature. Among the benign tumors found by Harrington and Moersch were cyst, hemangioma, mucocele, neurofibroma, papilloma, polyp, aberrant thyroid, myxofibroma, lipoangioma and adenoma. Recently Harrington has removed a large myxofibroma successfully.

Symptomatology. Symptoms of the presence of such a tumor are most often lacking.

Dysphagia is the most common, however, but even this is usually present only when the tumor mass becomes very large or a pedicle develops. Dyspnea because of tracheal pressure has been reported. Bleeding is very rare.

Diagnosis of the lesion is usually accomplished only at necropsy. The difficulty lies in the fact that so many of the tumors occur in the lower portion of the esophagus and are so often asymptomatic. With the aid of esophagoscopic examination it is possible to detect most lesions once their presence is suspected. This method of examination is not infallible, however, nor is roentgenographic examination, although both are of value in establishing a diagnosis.

Treatment. Treatment of the tumors when found varies with their size and location. It is best to attempt some type of extirpation of the lesion. Harrington and Moersch recommended esophagoscopic removal of the smaller, pedunculated tumors but suggested the surgical removal of tumors involving the muscular layers of the esophagus or tumors in which the attachment is not accessible.

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THE PRESENT STATUS OF THE SURGICAL TREATMENT OF CARCINOMA OF THE THORACIC ESOPHAGUS*

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THERE has been a renewed interest on the part of the surgical profession in recent years in the treatment of cancer of the esophagus. I believe the reason for this is the general dissatisfaction with the hit-and-miss therapy usually accorded this disease throughout the world. Until recently, the diagnosis of carcinoma of the thoracic esophagus carried with it a fatal prognosis and was followed by palliative treatment in the form of deep *x*-ray therapy with or without a permanent gastrostomy. The final mortality with this treatment was close to 100 per cent. Inasmuch as this was the generally accepted method of handling this disease for many years, a large experience accumulated and it is now possible to scrutinize the effect of *x*-ray therapy, especially from the standpoint of cure. An enormous literature on the radiation treatment of cancer of the esophagus has accumulated throughout the world. Of the vast number of cases reported, approximately twenty are said to be cured. Careful study of the published results in these cases, in the light of our present day concept of cancer, leaves many of them open to question because, in a considerable percentage, a positive biopsy was not reported, and in the remainder, the follow-up period was too short to warrant the contention that the disease was cured.

Except for an occasional recovery reported since Torek's original successful operation in 1913, the surgical treatment of cancer of the esophagus did not receive the impetus that it deserved until recent developments in the field of anesthesia, and until the physiological changes in the circulatory and respiratory systems accompanying thoracic operations were more clearly understood. With the latter there has been marked progress in the field of thoracic surgery. Surgeons now enter the thoracic cavity with the same sureness as they do when performing a laparotomy. The impetus to attack cancer of the thoracic esophagus by radical surgical means has been strengthened by a gradually increasing knowledge of the subject of cancer in general, and by a clearer understanding of the necessity of radically removing a cancer-bearing focus with all the lymphatic tributaries draining the particular site.

During the past four years, there has appeared, in the American literature especially, an increasing number of articles dealing with the surgical treatment of cancer of the thoracic esophagus. The most recent, that by Adams, published in the February, 1941 issue of *Surgery, Gynecology and Obstetrics*, attempts to summarize all of the cases accorded operative therapy. It is interesting to note that the fourteen patients successfully operated upon before 1934, died within two years after operation, with the exception of Torek's patient, who lived for thirteen years and died at the age of eighty-two of pneumonia. Between 1938 and 1940, thirty-four patients were successfully operated upon, and, of these, twenty or 58 per cent were living and well, some of them up to four years. During this period, about 50 per cent of the sixty-eight reported operations, were successfully consummated. In my opinion, Adams' report does not picture the situation as completely as I believe it to be. I am aware of at least ten other patients who were operated upon by surgeons in New York alone during the period mentioned by Adams, that were not incorporated in his paper. In

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addition, I know of other successful resections carried out in San Francisco, Washington, Boston and Philadelphia, which were not included in Adams' paper.

From a pathological standpoint, it is important to emphasize that cancer of the esophagus may remain a local disease for a considerable period of time. I would like to stress the fact that the life history of this disease in relation to the findings at operation is vastly different from the usual picture seen in the terminal stages on the autopsy table. Although extension to the regional lymph-nodes may occur late, the tumor itself may become locally inoperable because of fixation to vital structures such as the aorta, bronchus or vertebral column. It must be remembered that the length of history of dysphagia does not picture the extent or size of the tumor. Dysphagia, the outstanding symptom of cancer of the esophagus, does not occur until gross involvement of the esophageal lumen takes place. The tumor may grow outwardly rather than toward the lumen of the organ and may reach an inoperable stage before persistent dysphagia becomes a prominent symptom. A few years ago, I was under the impression that all patients with a short history could probably be classified as operable. But following a rather extensive experience with this disease since then, I have reached the opinion that too great stress should not be placed on the duration of the history in determining operability. I have seen a patient with a history of a year's duration, in whom a radical resection was carried out, and, on the other hand, I have noted three or four other patients with short histories averaging from two to four months, in whom exploration revealed inoperable tumors. In determining operability from the pathological standpoint, it must be emphasized that the location of the tumor is of greater importance. Experience indicates that those tumors located in the middle third of the organ between the arch of the aorta and the left main bronchus quickly become inoperable because of early fixation to one or the other structure. Tumors located in the superior mediastinum just above the arch of the aorta likewise become inoperable early in the course of the disease because these are prone to involve the left recurrent nerve and seem to spread more quickly to the nodes at the base of the neck. Fixation to the right mediastinal pleura does not, by itself, render the tumor inoperable. On four occasions, I have been able to excise a portion of the right mediastinal pleura with the growth and successfully bring the patients through the operative ordeal in spite of a bilateral pneumothorax on the operating table.

Cancers of the middle third of the esophagus (that portion situated from the upper surface of the aortic arch down to a point about 35 cm. from the upper incisor teeth) metastasize to the mediastinal lymph-nodes, and, late in the disease, to the supraclavicular nodes on the left side. Occasional retrograde metastasis may take place downward below the diaphragm to involve the nodes in the paracardial region and in the gastrohepatic omentum. Cancers located in the distal third of the organ, the most common site (that extending from the lower part of the middle third as far as the cardia of the stomach) usually spread to the regional nodes in the mediastinum. Not infrequently, there is extension below the diaphragm to the paracardial and gastrohepatic nodes. Involvement of the liver is, in my experience, a most unusual route of extension, except in the terminal stages of the disease. Inasmuch as the operation of choice today for cancers of the distal third is a transthoracic, transdiaphragmatic resection with anastomosis between esophagus and stomach, it is always possible to determine lymph-node extension below the diaphragm during the course of the operation. Because lymph-node extension below the diaphragm is most unusual with tumors located in the middle third of the organ, it is rarely necessary to explore the abdomen during the conduct of the operation for removal of tumors in this part of the organ.

It is important to emphasize the implications of the nature of the histological examination of the biopsy specimen obtained by esophagoscopy. The finding of squamous cell characteristics in the tumor indicates origin of the growth from the esophageal mucosa. If adenocarcinoma is found on histological examination, there is definite evidence that the tumor arises from mucosa of the cardia or upper portion of the stomach. I have heard clinicians and pathologists state that adenocarcinoma of the distal esophagus often originates from misplaced gastric mucosa in the esophageal wall. I believe this to be grossly inaccurate. A fairly large operative experience indicates that every instance of adenocarcinoma of the esophagus has demonstrated that the tumor originated in the cardia or upper portion of the stomach.

The lymph-node spread from adenocarcinoma of the cardia is mainly below the diaphragm. In the majority of instances, the extension is in the direction of the paracardial and gastrohepatic nodes. Occasionally, the peripancreatic nodes may become involved. Extension to the portal system with metastatic involvement of the liver may occur early in the disease. In a few instances, I have noted extensive node involvement in the mediastinum above the diaphragm with minimal involvement below the diaphragm. This clear distinction of the difference in lymphatic spread of squamous cell carcinoma and adenocarcinoma is the reason for my suggestion that every case of adenocarcinoma of the lower esophagus should be explored through an abdominal incision in order to determine operability.

Cancer of the esophagus occurs most frequently in the fifth and sixth decades of life. I have seen two patients in the third decade and many in the fourth. The disease apparently is more common in men than in women. There is nothing in any of the histories that I have elicited, which would indicate some predisposing cause, such as chronic irritation. Without exception, every patient has presented the one cardinal symptom of dysphagia, i.e., interference with the normal act of swallowing. Most often, this difficulty is concerned with the swallowing of solid foods and the patient usually states that swallowed food seems to stop at a point in the esophagus which the patient indicates by pointing to a portion of his sternum. Most of the patients have been quite accurate in localizing the site of obstruction, when this was compared with the *x*-ray films and the findings at operation. Pain on swallowing has not been a prominent symptom in the cases that I have seen. In almost every instance, dysphagia has been progressive, with gradual elimination of solid food from the diet. Most of the patients were able to subsist only on fluids at the time of the first examination.

The extent of loss of weight has been of considerable interest in determining the duration of the disease and arriving at an estimation of operability. My experience to date would seem to indicate that too much reliance should not be placed upon weight loss as an indication of the extent of the disease. The tumor which early impinges upon the lumen will produce a rapid weight loss early in the course of the disease. I have seen one patient who was known to have cancer of the esophagus for one year and yet there had been no weight loss whatever. By the same token, the extent of weight loss has not proved to be of any significance in arriving at an opinion concerning operability.

The symptom of persistent back pain is of serious importance. It usually indicates extension of the growth beyond the wall of the organ with fixation to the aorta or vertebral column. With adenocarcinoma of the cardia, backache may mean pancreatic extension. However, the symptom of backache does not necessarily indicate inoperability because it may be caused by inflammatory reaction in the peri-esophageal tissues.

Every patient with carcinoma of the esophagus should be carefully examined for the presence or absence of an enlarged

supraclavicular lymph-node, the so-called node of Virchow. The finding of a firm supraclavicular node demands biopsy of the node before attempting surgical excision of the esophageal tumor. If node metastasis is demonstrated on histological examination, the case should be classified as inoperable.

It is needless to stress the fact that every patient who complains of persistent dysphagia should be subjected to careful roentgen examination of the esophagus and upper stomach. A negative examination does not exclude the presence of a neoplasm of the organ. If the symptom of dysphagia persists in spite of symptomatic treatment, examination should be repeated after three or four weeks. The finding of an irregularity, a filling defect, or an obstruction, on fluoroscopic and *x*-ray examinations, calls for esophagoscopy by a competent operator. There seems to be an idea in the medical profession that esophagoscopy is a dangerous procedure. There is little argument with the fact that, in the hands of an inexperienced esophagoscopist, this is quite true. However, with a disease such as the one under discussion, it is important to verify by direct visualization and by biopsy examination, the presence or absence of a carcinoma before attempting radical surgical extirpation. Therefore, the alleged danger of the esophagoscopic examination itself has little significance when compared with the necessity of establishing a positive diagnosis.

Esophagoscopy has a twofold purpose: In the first place, the gross appearance of the tumor is noted and a biopsy specimen is taken to determine the histological characteristics. Secondly, the esophagoscopist notes the distance of the growth from the upper incisor teeth. This, in conjunction with the roentgen film, aids the surgeon in deciding the type of operation he will undertake. As stated before (and it is necessary to emphasize this point), the demonstration of adenocarcinoma in the biopsy specimen calls for preliminary abdominal exploration to determine operability because of the direction of lymphatic spread that occurs with this tumor. With tumors located in the middle third of the organ, one type of operation is indicated, while, with tumors in the distal third (beyond 35 or 36 cm. from the upper incisor teeth) another operative procedure is carried out.

The decision to undertake radical surgical extirpation of a cancer of the esophagus will rest on a number of factors. The age and general condition of the patient are of considerable importance. It would be poor judgment, in my opinion, to subject a patient in his seventies, with a poor heart, arteriosclerosis and poorly functioning kidneys, to the hazards of so extensive a procedure. On the other hand, I have successfully carried out a one-stage resection in a man of seventy-three years. Each case must be judged on its own merits and it is important to have the help of a competent physician to aid the surgeon in estimating the patient's physical and physiological status. The demonstration of metastatic involvement of the supraclavicular nodes indicates inoperability. Tumors located between the arch of the aorta and the left main bronchus present a special problem. Fixation to either or both structures takes place early and the technical difficulties resulting therefrom may be formidable. The outlook from the standpoint of cure, should the patient survive the operation, is not favorable. None of the four patients in my series has survived a year, and each has died of recurrence in the superior mediastinum. Fortunately, growths in this location are not frequent.

The duration of the history of dysphagia is of some importance in determining operability, but I do not, at the present time, consider it as important as I formerly did. Intralumenal extension of the tumor and the degree of fibrotic constriction of the circumference of the organ are important pathological reasons for the severity of symptoms. These must be carefully appraised.

CHART I

SQUAMOUS CELL CARCINOMA OF THE ESOPHAGUS

Pt.	Male	Female	Age	Approximate Duration of Symptoms	Location of Tumor, Cm.	Biopsy	Operation	Postoperative Complications	Follow-up	Remarks
M. A.	..	✓	54	8 weeks	36	Squamous cell ca.	9/18/39 resection with intrathoracic esophagogastrostomy.	None.	Alive and well 2 yrs.	Moderate stricture at anastomo. requiring occ. dilatation.
B. B.	..	✓	48	6 months	30	Squamous cell ca.	4/20/36 gastrostomy. 4/29/36 Torek.	Left empyema, Pellegra, recurrence in esoph. stump 6 mo. later.	Died of recurrence in superior mediastinum 22 mo. later.	First case of series.
S. B.	✓	..	50	3 months	26	Squamous cell ca.	9/1/39 Janeway gastrostomy. Tumor inop.	None.	Died four mo. later of disease.	
J. C.	✓	..	60	5 weeks	39	Squamous cell ca.	4/27/39 resection with intrathoracic esophagogastrostomy.	Right pleural effusion 1200 cc.	Alive and well 2½ yrs.	No evidence of stricture.
P. C.	✓	..	54	3 months	31	Squamous cell ca.	2/20/40 Witzel gastrostomy. 2/28/40 Torek.	Localized left empyema.	Died of mediastinal recurrence 11 mo. later.	Part of rt. mediastinal pleura excised at op.
F. C.	✓	..	65	4 months	25	Squamous cell ca.	7/28/36 Janeway gastrostomy. 8/27/36 Explor. inop. supra-aortic.	Lobar pneumonia.	Died of disease 2 mo. later.	
A. G.	✓	..	62	5 weeks hoarseness 3 days	24	Squamous cell ca.	5/26/38 Janeway gastrostomy. 6/13/38 Explor. inop. supra. aortic.	Fulminating pneumonia.	Died on 6th postoperative day of pneum.	
M. G.	✓	..	35	8 weeks	30	Squamous cell ca.	8/21/39 Janeway gastrostomy. 8/31/39 Torek. Tumor bet. bronchus and aorta.	R.L.L. pneumonia, encapsulated left empyema.	Died 1 year later of mediastinal recurrence.	
M. McC.	✓	..	61	3 months	32	Squamous cell ca.	9/16/39 Janeway gastrostomy. 9/30/39 Torek.	Cerebral hemorrhage.	Died 2nd day postop.	
H. M.	..	✓	53	8 weeks	34	Squamous cell ca.	11/16/36 Janeway gastrostomy. 11/28/36 Torek. 6/24/37 Antethoracic esoph.	None.	Alive and well 5 yrs.	Second case of series.
E. P.	✓	..	56	10 weeks	34	Squamous cell ca.	3/14/39 Janeway gastrostomy. 3/22/39 Torek. Piece of rt. mediastinal pleura excised.	Tension pneumothorax, right.	Died 2nd day postoperative.	Death due to error of judgement.
F. S.	✓	..	49	3 weeks	34	Squamous cell ca.	10/26/39 Resection with intrathoracic esophagogastrostomy.	Surgical shock.	Died first day postop.	Exceedingly difficult technical procedure.
J. S.	✓	..	47	7 months	30	Squamous cell ca.	10/10/38 Janeway gastrostomy. 10/20/38 Torek. Tumor bet. bronchus and aortic arch.	None.	Intensive radiation therapy postop.	Died one yr. later of mediastinal recurrence.
J. T.	✓	..	48	5 weeks	35	Squamous cell ca.	9/13/37 Janeway gastrostomy. 9/20/37 Torek.	Unexplained tem. to 106 on second day.	Died 3 mo. later of coronary disease.	Autopsy showed cav. in mediast. No cancer tissue.

CHART I (*Continued*)

Pt.	Male	Female	Age	Approximate Duration of Symptoms	Location of Tumor, Cm.	Biopsy	Operation	Postoperative Complications	Follow-up	Remarks
G. B.	..	√	43	3 months	26	Squamous cell ca.	7/14/41 thoracic exploration. Tumor inoperable Stamm gastrostomy.	None.	Still alive.	
S. F.	√	..	59	4 weeks	39	Squamous cell ca.	5/8/40 resection with intrathoracic esophagogastrostomy.	Postoperative pneumonia of both lungs.	Died 3rd day postop.	Autopsy showed involvement of peripancreatic and coeliac nodes.
J. K.	√	..	53	2 months	30	Squamous cell ca.	4/3/40 Spivack gastrostomy. 4/17/40 Exploration. Inop. tumor.	None.	Died four mo. later of disease.	
R. F.	..	√	43	4 months	30	Squamous cell ca.	7/29/40 modified Torek preserving distal 2 inches of organ.	Unexplained convulsion on third day.	Alive and well 1 yr.	
H. K.	√	..	66	10 weeks	29	Squamous cell ca.	7/1/40 Janeway gastrostomy. 7/24/40 Exploration. Inop. tumor attached to aorta.	None.	Died four mo. later of disease.	
R. L.	√	..	67	4 months	33	Squamous cell ca.	5/11/40 Spivack gastrostomy. 5/29/40 Torek.	R.L.L. Bronchopneumonia. Pulmonary edema.	Died 48 hrs. postoperative.	
B. A.	..	√	57	1 year	29	Squamous cell ca.	11/23/40 modified Torek.	Cerebral embolus. Left empyema.	Died 11th postop. day.	Long hist. no wt. loss. Resectable tumor.
W. B.	√	..	72	10 weeks	36	Squamous cell ca.	1/8/41 resection with intrathoracic esophagogastrostomy.	Small right pleural effusion.	Alive and well 8 mos. postoperative. No stricture.	Part of rt. mediastinal pleura excised. Age of pt. noteworthy.
S. G.	√	..	52	4 weeks	30	Squamous cell ca.	3/20/41 modified Torek.	Developed fulminating pneum.	Died on 9th postop. day.	Operative risk not good.
W. M.	√	..	54	7 weeks	33	Squamous cell ca.	4/30/41 resection with intrathoracic esophagogastrostomy.	None.	Alive and well 5 mos.	There was also an early adenoca. of stomach near cardia. This was also excised.
Newark case.	√	..	53	5 months	30	Squamous cell ca.	4/10/39 Janeway gastrostomy. 4/19/39 Exploration—inoperable.	None.	Died 3 mos. later of disease.	
M. G.	√	..	64	3 months	20	Squamous cell ca.	4/19/41 cervical exploration. Inoperable. 6/2/41 gastrostomy.	None.	Still alive.	Left recurrent nerve paralysis.
G. R.	√	..	49	6 months	29½	Squamous cell ca.	12/18/40 modified Torek op.	Acute coronary occlusion with infarction of myocardium.	Died 3d day postoperative.	Cardiac death.
S. S.	..	√	56	3 months	29	Squamous cell ca.	9/7/40 thoracic exploration. Inoperable. Witzel gastrostomy.	None.	Died 3 months later.	Tumor adherent to aorta and pericardium.
P. K.	√	..	58	3 months	37	Squamous cell ca.	11/27/40 abdominal exploration. Inoperable. Gastrostomy.	None.	Died 4 mos. later of disease.	Tumor adherent to aorta and vertebral col.
J. K.	√	..	42	6 months	33	Squamous cell ca.	11/27/40 thoracic exploration. Inoperable. Stamm gastrostomy.	None.	Died 3 mos. later of disease.	Tumor adherent to thoracic aorta.

CHART II

ADENOCARCINOMA OF THE CARDIA

Pt.	Male	Female	Age	Approximate Duration of Symptoms	Location of Tumor, Cm.	Biopsy	Operation	Postoperative Complications	Follow-up	Remarks
H. Z.	√	..	62	5 months	40	Adenocarcinoma.	10/13/39 abdominal exploration. Inop.	None.	Died of disease 4 mos. later.	
L. S.	√	..	36	6 months	39	Adenocarcinoma.	10/19/38 thoracic exploration. Inoperable. Gastrostomy.	Fulminating pneumonia.	Died on 3d day postop.	At present time would explore abdomen first to determine operability.
M. M.	√	..	60	4 weeks	40	Adenocarcinoma.	10/6/39 transthoracic exploration. Inop. Gastrostomy.	None.	Died 5 mos. later.	At present time would explore abdomen first to determine operability.
S. R.	..	√	58	6 weeks	38	Adenocarcinoma.	5/21/40 abdominal exploration. Transthoracic resection with anastomosis.	None.	Well 16 mos. postoperative.	One lymph-node showed involvement.
A. M.	√	..	60	about 9 months.	40	Adenocarcinoma.	6/6/40 abdominal exploration. Transthoracic resection with anastomosis.	None.	Well 15 mos. postoperative.	No involved nodes found.
C. L.	√	..	60	2 months	39	Adenocarcinoma.	3/7/40 abdominal exploration. Inoperable. Spivack gastrostomy.	None.	Died of disease 3 mos. later.	
H. S.	√	..	67	2 months	40	Adenocarcinoma.	6/28/40 abdominal exploration. Inoperable.	None.	Died 4 mos. later of disease.	
S. L.	√	..	48	10 weeks	35	Adenocarcinoma.	12/28/40 abdominal exploration. Inoperable.	None.	Died 5 mos. later of disease.	
G. K.	..	√	55	6 months	38	Adenocarcinoma.	10/25/40 abdominal exploration. Inoperable. Janeway gastrost.	None.	Died 3½ mos. later of disease.	
I. G.	..	√	52	8 weeks	39	Adenocarcinoma.	6/28/40 abdominal exploration. Inoperable. Janeway. gastrostomy.	None.	Died 6 mos. later of disease.	
J. G.	√	..	46	6 months	40	Mucus cell carcinoma.	3/10/41 abdominal exploration. Transthoracic resection and anastomosis.	None.	Well 6 mos. postoperative.	Associated lymph-nodes removed with tumor showed involvement.
H. C.	√	..	66	1 year	40	Adenocarcinoma.	4/1/41 abdominal exploration. Transthoracic resection with anastomosis.	Coronary occlusion on 6th day.	Died suddenly on 6th day.	Length of history and operability noteworthy.
I. L.	√	..	51	4 weeks	40	Adenocarcinoma.	5/5/41 abdominal exploration. Inoperable.	None.	Still alive.	

CHART II (*Continued*)

Pt.	Male	Female	Age	Approximate Duration of Symptoms	Location of Tumor, Cm.	Biopsy	Operation	Postoperative Complications	Follow-up	Remarks
J. A.	✓	..	49	3 months	39	Adenocarcinoma.	1/26/39 transthoracic resection and anastomosis.	Cerebral embolus.	Died on 3d day.	Lymph-nodes showed involvement.
M. B.	..	✓	70	8 months	39	Adenocarcinoma.	1/29/38 abdominal exploration. Inoperable. Janeway gast.	None.	Died of disease 2 mos. later.	
J. B.	✓	..	62	4 months	40	Adenocarcinoma.	10/5/39 abdominal exploration. Inop. Janeway gast.	None.	Died of disease 3 mos. later.	
W. C.	✓	..	47	3½ months	39	Adenocarcinoma.	10/25/39 abdominal exploration. Chest exploration. Inoperable.	None.	Died of disease 4 mos. later.	Extensive mediastinal node involvement.
C. D.	✓	..	56	5 months	36	Adenocarcinoma.	2/1/38 Janeway gastrostomy. 4/19/38 chest exploration. Inoperable.	Postoperative pneumonia.	Died one mo. later.	
L. E.	..	✓	73	10 weeks	39	Adenocarcinoma.	8/3/39 transthoracic resection and anastomosis.	Became comatose on 3d day.	Died on 4th day.	Autopsy essentially negative.
W. H.	✓	..	49	6 weeks	40	Adenocarcinoma.	6/20/39 transthoracic resection and anastomosis. 7/2/39 right thoracotomy for pyopneumothorax.	Developed lung abscess, ruptured of rt. lung.	Lived for 16 mos. Died of extensive retroperitoneal metastases.	Resected specimen showed extensive node involvement.
H. H.	✓	..	39	6 weeks	40	Hodgkin's disease of cardia.	4/12/41 abdominal exploration. Transthoracic resection and anastomosis.	None.	Doing well 5 months postoperative.	This case is included in series for obvious reasons.
M. F.	✓	..	61	5 months	40	Negative biopsy.	5/14/41 exploratory laparotomy. Inop.	Several severe gastric hemorrhages.	Still alive.	
M. B.	✓	..	57	3 months	40	Adenocarcinoma of cardia and lower esophagus.	5/21/41 exploratory laparotomy. Transthoracic resection and esophagogast.	Mediastinal abscess and peritonitis.	Died on 9th day postoperative.	Leakage from small perforation of esoph. at site of operative injury. Suture line intact.
H. G.	✓	..	57	3 months	40	Adenocarcinoma.	6/4/41 abdominal exploration. Inop.	None.	Still alive.	

Once the decision to undertake surgical treatment is made, the patient's general condition and his physical reserve after thorough preoperative preparation must be carefully appraised. It is important to pay special attention to oral hygiene. Carious teeth should be extracted and infected gums should be cleansed. This will naturally decrease the possibility of postoperative pulmonary complications. Great weight loss may be due primarily to extreme dehydration. The daily intravenous

injection of 1,000 to 2,000 cc. of 5 per cent glucose in saline over a week's period often results in astonishing improvement of the patient. In a recent case, the patient gained ten pounds in seven days. Unless complete obstruction is present, the patient should be encouraged to take large quantities of high-caloric liquids containing the necessary vitamins and minerals. In many instances, parenteral administration of vitamins will be necessary. It will be found that taking one or two weeks to improve the patient's general condition is time very well spent.

The question of preliminary gastrostomy deserves special mention. Till recently, I was of the opinion that most patients required a preliminary gastrostomy to aid in restoring fluids, proteins, etc. At the present time, I have the opposite viewpoint and believe that, except for the rare exception, most patients can be gotten into satisfactory condition by the methods above enumerated. To aid in restoring lost proteins, I have, in the last few patients, given amino acids intravenously, after the method of Elman. I believe this will prove to be of inestimable value in the preoperative preparatory period. The presence of a gastrostomy would seriously interfere with the carrying out of the operative procedure I recently described for carcinoma of the middle third of the organ and with the operation of resection and esophagogastrostomy for tumors of the distal third. Should alimentary feeding be necessary, jejunostomy instead of gastrostomy should be performed, and the operative incision should be placed at or below the umbilical line.

In most instances, it will be necessary to administer a blood transfusion before operation. A transfusion *during* operation is, in my opinion, obligatory. A third transfusion during the early period of convalescence is also most desirable. In order to decrease the number and virulence of the organisms covering the ulcerated surface of these neoplasms, I have routinely administered one of the sulfonamide drugs (usually sulfanilamide) for seventy-two hours prior to operation, in 15 gr. doses every four hours. An additional line of attack against the bacterial flora of the ulcerated neoplasm has consisted of mechanical cleansing by frequent irrigation with warm saline solution through a Levin tube.

A few writers have emphasized the need of pneumothorax prior to operation in order to accustom the lung to the positive intrapleural pressure which occurs during the operation. I have never seen the need for this, relying on the anesthetist to maintain varying degrees of positive intrapulmonary pressure. I do not subscribe to the recommendation of a few authors that the phrenic nerve be crushed either prior to or during the operation. Paralysis of the diaphragm adds another burden to an already handicapped patient in his effort to obtain maximum pulmonary ventilation.

In my experience, the combination of avertin and cyclopropane or ethylene has been most satisfactory. Complete collapse of the lung should not be permitted to take place at any time and the anesthetist should have complete control of the situation. It is important to secure the services of a competent anesthetist because the incidence of postoperative pulmonary complications depends in large measure on the skill of administration of the anesthetic agent.

A few writers recommend intratracheal anesthesia, stating that the degree of inflation of the lungs can be more accurately controlled by this method. I have believed that the trauma of intubating the trachea in these debilitated patients may well be a factor in the development of pulmonary complications and have, therefore, had no experience with this method.

The ideal to be sought after in the surgical treatment of cancer of the thoracic esophagus is radical resection of the cancer-bearing portion with all the associated lymph-nodes and re-establishment of esophagogastric continuity. With tumors located in the middle third of the organ, it has been impossible, up to the present time,

to attain this ideal to its fullest extent because, with the operative procedures so far evolved, it has been impossible to re-establish continuity following a radical resection. Although the Torek operation has stood the test of time and experience and although I have modified it to the extent of eliminating a preliminary gastrostomy and of preserving the sphincteric action of the cardia, I have always believed it was a makeshift procedure, leaving the patient with an artificial esophagus which was far from desirable from all standpoints. Because of this, I am trying to develop a new operation which will eliminate these objections. I am not prepared to say what the final result will be but the procedure seems to have some merit.

With tumors located in the distal third of the organ or in the cardiac end of the stomach, the operation of choice is a left transthoracic transdiaphragmatic resection of the lower esophagus and upper stomach, mobilization of the proximal two-thirds of the stomach and a suture anastomosis between the end of the severed esophagus and the upper anterior wall of the stomach. In previous papers, I have described in great detail, the technical steps of this operation and also, of the modified Torek procedure for cancers of the middle third. That the operation of esophagogastrostomy is physiologically and anatomically sound is proved by the fact that there has not been a single instance of leakage from the suture line in the entire series of cases reported herewith. In a recent case, post-mortem examination disclosed leakage from a perforation of the esophagus one-half inch above the suture line due to operative injury of the organ at this site, a preventable technical error.

It must be emphasized that the operation for cancers of the middle third carries with it a greater postoperative morbidity and a higher mortality than the operation of esophagogastrostomy for tumors of the distal third. Greater operative trauma involving the entire mediastinum instead of only the distal portion, handling of the arch of the aorta in freeing the esophagus from its posterior surface and the possibility of increasing the already existing pneumothorax during the cervical phase of the operation are major reasons for the increased morbidity and mortality. In spite of the fact that the Torek operation is an excellent one from the standpoint of free exposure and good cancer surgery, the above enumerated objections have made for dissatisfaction and a desire to provide a method which would approach the ideal already expressed.

My own experience to date consists of the operative treatment of fifty-four cancers of the esophagus and cardia. The accompanying chart indicates the important features of each case history. A summary of the entire group shows that of thirty squamous cell carcinomas of the esophagus, nineteen were considered operable and were resected, an operability rate in this group of 63.3 per cent. In the group of nineteen resections, there were eight postoperative deaths, an operative mortality of 42 per cent. The causes of death are listed in the chart. There were thirteen Torek or modified Torek operations with six deaths and six anastomosis operations with two deaths, with a comparable mortality rate of 46 and 33.3 per cent, respectively. In the Torek group, only two patients are alive and well today, one almost five years and the other thirteen months postoperatively. In the anastomosis group of four operative survivors, all are alive and well, two and one-half years, two years, eight months, and five months, respectively.

In the group of adenocarcinoma of the cardia with esophageal involvement, there were twenty-four cases. Nine were found operable and subjected to radical resection and anastomosis. The operability rate was 37.5 per cent. There were four operative deaths, a mortality rate in the adenocarcinoma group of 44.4 per cent. Of the five operative survivors, four are alive and apparently free of disease sixteen months, fifteen months, six months, and five

months, respectively. The fifth patient died of retroperitoneal recurrence one and one-half years after operation.

If we combine the adenocarcinomas of the cardia and the squamous cell tumors of the distal esophagus into one group, it will be seen that the operation of transthoracic resection with esophagogastrostomy was carried out in fifteen cases. There were six deaths, an operative mortality of 40 per cent. Eight of the operative survivors are alive and well for periods ranging from two and one-half years to five months.

The general problem of the surgical treatment of cancer of the esophagus may, on the basis of the experiences above described, be reduced to cold figures. The average patient with a cancer of the middle third of the organ stands a 54 per cent chance of surviving the operation and a 30 per cent chance of living more than one year. With squamous cell cancer of the distal third of the organ, the patient has an approximate 70 per cent chance of surviving the operation and almost 100 per cent probability of living more than two years. In the group of adenocarcinoma of the cardia, the probability of operability is only about 37 per cent. There is a 60 per cent chance of operative survival and an 80 per cent probability of living more than one year.

Such is the present status of the operative treatment of this disease. The figures presented indicate a great advance over those given heretofore for the treatment of this disease by other methods. However, there is much to be desired. I am firmly convinced that the operability percentage, the mortality rate and the postoperative survival period will be markedly improved when more early cases are referred for operation and with increasing experience in the operative management.

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RESUSCITATION FOR CARDIAC STANDSTILL AND VENTRICULAR FIBRILLATION OCCURRING DURING OPERATION*

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THIS discussion concerns the resuscitation of patients who die in the operating room. The type of death under discussion is that which occurs unexpectedly. It occurs in the patient who possesses normal heart and lungs. Its occurrence in a healthy patient is not unlike turning off the ignition in a good motor. A vital spark is lost in an otherwise good mechanism. Fortunately, this type of death does not occur frequently. When it does occur, however, the stake is high. I can recall several instances in my own experience. The first one happened twenty years ago when I was a surgical intern in one of the hospitals in Baltimore. A pulmotor was brought to the operating room by the fire department. The patient could not be revived. Unsuccessful attempts at resuscitation take place in the operating rooms the world over. My belief is that surgeons should not turn these emergencies over to the care of the fire department. We should take care of them ourselves. To do so requires a program of action established before the emergency occurs and put into use as soon as the emergency appears. Training in resuscitation should become a part of the surgical curriculum.

In order to take care of these emergencies we must have a proper appreciation of the oxygen requirements to sustain cell life for which an almost constant supply of oxygen is necessary. The supply of oxygen can be interrupted for only a few minutes and then the flame of life goes out. Life can be restored if the interruption in the oxygen system is brief. The safe period for the human being is not definitely known but animal experimentation places it at three to five minutes.[1] The respiratory center rapidly loses its function if it is deprived of oxygen for longer periods of time. If respiratory function can be restored later on, resuscitation is possible. If it cannot be restored later on, then, of course, full recovery is impossible even though the heart action has been restored. Effective effort must be made to re-establish the oxygen system before respiratory loss becomes permanent. There is no place here for trial and error experimentation. When the emergency arises, proper steps must be taken without deliberation and delay.

The Oxygen System. The oxygen system includes respiration and circulation. (Fig. 1.) These two components are interdependent in transporting oxygen to the living cell. Whether respiration fails first or whether circulation fails first is of little consequence in resuscitation. The loss of either is equally destructive to the supply of oxygen to the cell and to cell life.

Respiration. The lungs must be ventilated properly for successful resuscitation. The methods of Schaefer and of Sylvester are valuable for resuscitation from drowning and can be used anywhere in the open country. However, these boy-scout methods of chest compression do not meet the demands of resuscitation in the operating room. Likewise the Drinker respirator is not satisfactory for this purpose. A constant stream of oxygen delivered into the lungs through an intratracheal catheter does not provide satisfactory ventilation of

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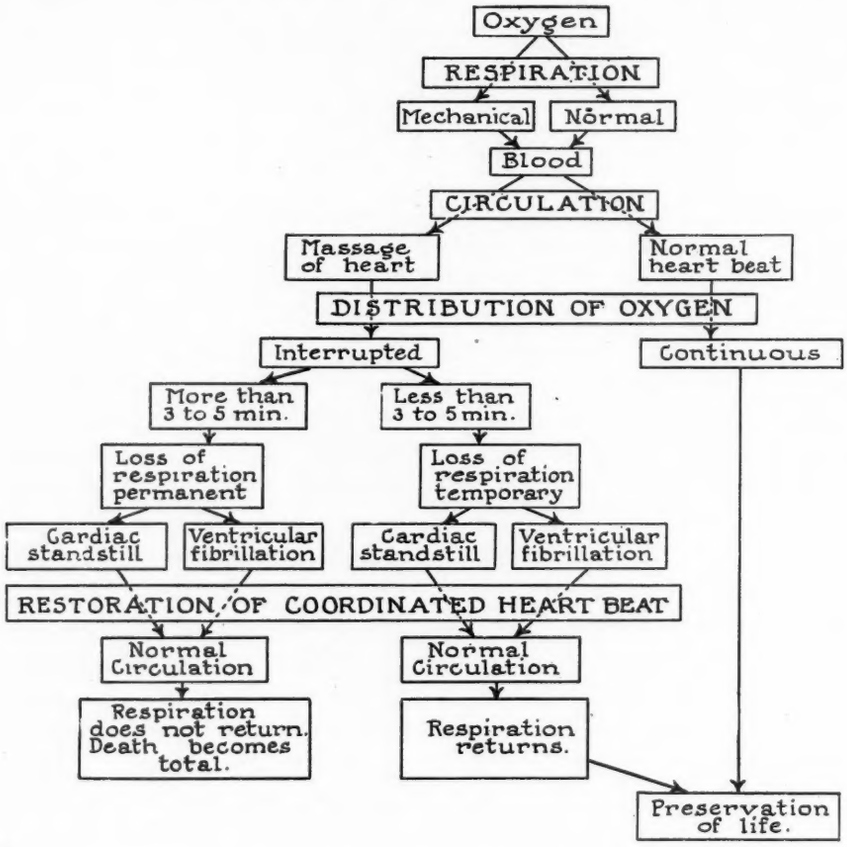
the lungs. Intermittent compression of a gas bag by hand does not meet the requirements. The only satisfactory method is one which intermittently fully distends the lungs and collapses the lungs and which operates in such a way that the surgeon can drape an operative field, open the chest and provide circulation by massage of the heart. For many years these requirements were met in experimental laboratories by several types of apparatuses which intermittently inflated and deflated the lungs. In our laboratory we used an intermittent current of air passed through an ether bottle and delivered into the lungs through an intratracheal tube. A windshield wiper device was used to make the current of air intermittent. A vent was placed in the tube to allow the lungs to deflate without obstruction. The volume of air sent in and the rate were easily regulated. Thousands of dogs were anesthetized in this way; the chest was opened and intrathoracic operations were carried out. *On the basis of this experience we can say that mechanical respiration if properly adjusted performs the function of pulmonary ventilation more thoroughly than does normal respiration.*



OXYGEN AND RESUSCITATION

FIG. 1. This chart indicates the transfer of oxygen from the outside air to the inside structures of the body.

A few years ago I began to use this apparatus on patients with increased intracranial pressure. I have carried several patients through brain tumor operations with this type of mechanical respiration after the respiratory center was thrown out of function. The introduction of mechanical respiration into our hospitals has been slow. There are few hospitals that possess an apparatus that can provide respiration if failure should occur during an operation on the brain, chest or abdomen. There are, indeed, few surgeons who appreciate the need for perfect mechanical respiration and

for purposes of resuscitation the respiration must be perfect. I learned what constitutes satisfactory pulmonary ventilation from my experience in resuscitating dogs. Without this experience I would not have learned this lesson.

There are several forms of apparatuses that can provide perfect mechanical respiration. Crafoord[2] and Mautz[3] each have devised mechanical respirators that meet the requirements. The Mautz apparatus, with which I am familiar, is simple and practical. (Fig. 2.) It is a closed system and uses a gas bag which is part of the equipment of the standard anesthetic machines in common use. The gas bag is enclosed in a glass box. A mechanical device forces air into the box which in turn compresses the gas bag and this in turn expels its contained oxygen or anesthetic mixture into the lungs. The pressure in the glass container is then released automatically and the gas returns from the lungs to the rubber bag. Soda lime absorbs the carbon dioxide. A

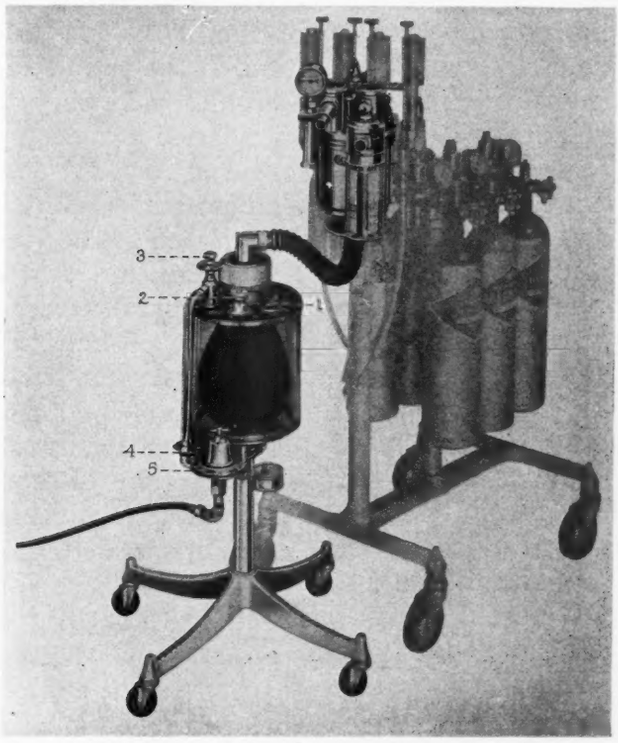


Fig. 2. The Mautz respirator, a mechanism for the intermittent compression of the breathing bag. The tube beneath the glass chamber connects to an air pressure line. This feeds air to an interrupter which works automatically and delivers air into the chamber under pressure and then allows air to escape from the chamber. The gas bag is thereby automatically compressed and delivers its contained gases into the lungs. A container of soda lime in the circuit absorbs the carbon dioxide. The gas bag can be filled with oxygen or other gases as desired. 1 is the valve which connects or disconnects the intermittent pressure by closing the chamber or opening it to the atmosphere; 2 is the pressure-limiting valve which accurately limits the pressure applied to the breathing bag; 3 is the rate control; 14 to 28 alternations per minute; 4 is a windshield wiper device which intermittently opens and closes valve in the bottom of chamber and regulates the alternations when valve 1 is closed. 5 is the reducing valve to regulate the air pressure to 4 lb. per sq. in., the pressure for which the device is calibrated.

pressure of 12 to 14 mm. of mercury will expand the lungs nicely. It is not always necessary to use an intratracheal tube. Crafoord emphasized the importance of free, unobstructed expiration in order to get rid of carbon dioxide. He pointed out that retention of carbon dioxide produces cardiac dilatation and failure even though plenty of oxygen is delivered to the lungs. He also discussed the fact that the machine takes over respiration and rests the muscles of respiration. This reduces the amount of oxygen consumed by the patient.* If the Mautz or Crafoord apparatus is not available, I would suggest that the inexpensive type of wind shield interrupter be used. I believe that every operating room should have available an apparatus that provides satisfactory mechanical respiration. To have it in working condition and to have it correctly adjusted the apparatus should be used almost routinely. We use it freely on patients when there is no need for it because we desire our anesthetists to be familiar with it.

When to Use Mechanical Respiration. It is obvious that respiration should be taken over by the machine as soon as evidence of poor ventilation appears. As a rule there is a warning before the oxygen system breaks down completely. A critical point is reached when the respiration becomes shallow and irregular and when the heart beat begins to lose its force. If good ventilation can be given at this point, the crisis sometimes can be averted. Much depends upon the supply of oxygen to the heart. Repeatedly in experimental work with the chest open we have observed the heart to recover its strength immediately after the lungs were properly blown up. Anoxemia and accumulation of carbon dioxide weaken the heart action and produce failure. Conversely, the heart beat is strengthened when plenty of oxygen is supplied and the carbon dioxide is removed.

It is also obvious that getting the oxygen in is of no value if there is not any circulation. If the circulation has disappeared, it becomes necessary to provide artificial circulation by massage of the heart. Mechanical respiration and circulation by cardiac massage are equally necessary to get oxygen to the vital brain cells.

Circulation. When the heart stops beating, the organ either comes to a complete cessation of activity or the ventricular muscle fibers undergo fibrillary twitchings for a variable period of time and then these fibrillary twitchings disappear. The method of restoration of the heart beat is not the same in each of these conditions and it is necessary to determine which condition is present before attempting to restore the beat. The first requirement, however, is not restoration of the beat but the moving of oxygen into the brain. This must be done before the brain centers disintegrate. As soon as the heart stops, action must be taken by the surgeon or his assistant. The chest is rapidly prepared for operation. An incision is made between two ribs over the precordium and the chest is opened. Costal cartilages, one above and one below, are cut across. A retractor is placed. The pericardium is widely opened. A hand is hurriedly placed on the heart and the heart is emptied of its blood. Active massage, about forty times a minute, is carried out. When this point has been reached the crisis has been passed and the need for quick action no longer exists. After a few minutes the operator observes the heart to determine whether or not it is fibrillating. The lungs are inspected to make sure that they are coming well up with each inspiration and that the deflation is also satisfactory. If all is well, the heart muscle becomes pink and regains its tone. It may start beating. If it does not start beating, restoration of the beat is the next problem to be undertaken. To do this some special training or experience is helpful. If a member of the

* One can appreciate the energy expended in breathing by entering the chamber devised by Dr. Alvan Barach of the Medical Center, New York.[4] In this chamber the pressure alternately rises and falls. When one enters this chamber the respiratory effort ceases and respiration is taken over by the chamber. The degree of relaxation and rest that one experiences in this chamber is remarkable.

staff possesses this experience, time can be taken to call him for this task.

Ventricular Standstill. Good ventilation of the lungs before the heart stops beating frequently will avert this type of failure. After the heart stops beating, pulmonary ventilation is not effective because oxygen is not transmitted from the lungs. Epinephrine injected into the heart through the chest wall might start it beating. The needle prick of the heart might produce a contraction. Compression over the precordium is sometimes effective. But these steps have usually been taken before the heart has actually stopped and time is not to be taken in waiting to see whether the heart will start again. The heart is exposed for massage.

If it does not start with direct massage, a small dose of epinephrine is injected into the blood stream. We use 1 cc. of a 1 : 1000 solution of epinephrine diluted in 10 cc. of sodium chloride solution and inject it into the cavity of the right ventricle or auricle. Massage is continued. This dose may be repeated. If the lungs are well ventilated, the heart will always start beating. The body should be kept warm with external heat. The heart should not be allowed to cool and warm saline is dripped upon it. If the brain revives immediately, an anesthetic may be necessary while the wound is being closed.

Ventricular Fibrillation. As a rule ventricular fibrillation cannot be made to disappear by massage and the use of epinephrine. In our experience the most effective method consists in the use of procaine followed by an electric shock. Five cc. of 2 per cent procaine are injected into the cavity of the right auricle or right ventricle. Massage of the heart moves the drug through the lungs, left side of the heart, aorta, coronary arteries and myocardial bed. The effect of the drug upon the myocardium is a reduction in its tone and a reduction in its irritability. The heart feels more flabby in the hand and it also dilates after the use of the drug. Large doses should not be used. Two large electrodes of metal are placed one on each side of the heart and a shock of one to one and one-half amperes is sent into the myocardium. The current is ordinary electric light current with sufficient resistance in the current to reduce the amperage. The current is made and then broken after a fraction of a second. The current brings the muscle fibers into a state of contraction. When they relax they may not show any movement. Not infrequently the fibers go back into the state of fibrillation and the shock must be repeated. If the heart is flabby and shows no inclination to contract, 1 cc. of epinephrine is used. This increases the tonus and the heart can be felt writhing in one's hand. The heart is again shocked. It ought to start if conditions are right. If it does not start to beat, the lungs are inspected to make certain that they are properly ventilated. We have also used 1 per cent calcium chloride in doses of 5 cc. injected into the cavity of the right ventricle. Calcium chloride and epinephrine each increases the tone of the heart muscle, reduces the degree of dilatation and stimulates the fibers to contract. These drugs also tend to preserve the state of fibrillation but this is not always true. We are cautious in the use of these drugs while fibrillation persists. However, we use either epinephrine or calcium chloride if the heart shows no disposition to beat after the use of procaine and electric shock.

The heart can be defibrillated. These methods are effective. It may require several attempts but if conditions are proper, such as the amount of oxygen, the tonus of the heart muscle and the irritability, the co-ordinated beat can be restored.

While all this is being done the body must be kept warm by external heat. Mechanical respiration is continued as long as necessary.

Results. Numerous reports of successful resuscitation are in the literature. These were in patients in whom ventricular standstill was present. In so far as I know the only successful attempts to defibrillate the human heart were in two patients by Beck.

In each of these patients the respiratory center was dead and did not revive. A co-ordinated heart beat was restored in each of these patients but the patients were lost because vital centers had disintegrated before the oxygen system was restored.

CASE REPORTS

Case 1. E. J., a Negro boy, aged nine years, had sustained an injury to the knee. An operation was done to remove a fragment of bone from the knee joint on December 7, 1938. The anesthetic was nitrogen monoxide and ether. The patient was kept in a light zone of anesthesia throughout. At 9:15 the skin was being sutured and the operation was practically completed. The respiratory rate was 19 per minute. At 9:20 the pulse suddenly became weak. Two minutes later it was impossible to get the blood pressure and the respiratory rate was 4 per minute. At 9:25 the pulse could not be felt. The pulse disappeared before respiration stopped. An intratracheal tube was inserted after respiration ceased, and mechanical respiration was given by the respirator. I came on the scene at about 9:32. Dr. J. A. Clark, the surgeon, stated that the pulse had been absent for about eight or nine minutes. This agrees with the time given by the anesthetist. The preparation of the field was done by Dr. Clark while I put on gloves and a gown. It required a few seconds to open the chest. The heart was massaged several times with the pericardium intact. The pericardium was then opened, and the heart was observed. The ventricles showed a coarse, slow type of fibrillation. The heart was dilated but time was not taken to determine the degree of dilatation. Rhythmic compression and relaxation of the heart by hand was done for about twenty minutes. This mechanical circulation produced a palpable pulse over the temporal artery. Never did the wink reflex return. The pupils were dilated and failed to react. Heat was applied to the body. Five cc. of 2 per cent procaine hydrochloride was injected into the cavity of the right auricle. The circulation was kept up by the hand. Large electrodes were placed on the ventricle, and a shock of about 1.5 amperes was sent through the heart. Three shocks were used. They produced forceful jerks of the body. The fibrillation stopped after these shocks. The heart was in standstill. The heart muscle was flabby in the hand and was without normal tonus. Epinephrine hydrochloride was injected into the blood through the right auricle and massage was continued. The heart remained flabby. Five cc. of 1 per cent solution of calcium chloride was injected. The heart remained flabby. It was then noted that the lungs were not properly inflated and that the tube was not in the trachea. It is probable that it slipped out when the patient jerked with the application of the electrical shock. The tube was inserted in the trachea and the lungs were properly expanded. The interval between the shocks and the discovery that the tube was out of the trachea was about ten minutes. There were a few moments of good aeration and then the heart started up in a forceful rhythmic contraction of 70 per minute. This occurred at 10:12, forty-seven minutes after the pulse disappeared. The radial pulse was easily palpable. The heart was observed for twenty minutes, and the incision in the chest was closed. The pulse later became weaker. There was no reflex at any time. The pulse disappeared at 12:00 o'clock. Necropsy showed no anatomic cause of death.

Case 11. F. B., a Negro boy, thirteen years old, had ptosis of the right eyelid following an injury. Transplant of fascia lata for correction of the ptosis was carried out on July 27, 1939. The anesthetic was nitrogen monoxide and ether. As the last silk suture completing the operation was about to be placed, the operator was informed by the anesthetist that respiration was not normal. At 9:22 respiration had ceased; the pulse rate was 30 per minute. At 9:26 the pulse could not be obtained. An airway was inserted into the pharynx. Epinephrine hydrochloride was injected into the heart. The patient was placed in a Drinker respirator, but it was impossible to expose the heart and provide circulation with the patient in this respirator. He was taken out and given mechanical respiration by means of the Mautz respirator. At 10:05 the heart was exposed by Dr. John Thornton, who at 10:09 began to move the blood by hand. The tone of the heart improved, and for forty-five seconds he obtained what he thought were weak co-ordinated contractions. These were replaced by fibrillary movements. Dr. Thornton sent a current through the heart but it did not stop the fibrillation. Dr. Thornton continued to produce circulation by manual massage of the heart. At approxi-

mately 10:45 I was asked whether I would like to try to defibrillate the ventricles of the heart. I found coarse fibrillary movements of the ventricles; it was obvious to any one that the ventricles were fibrillating. The manual emptying of the heart expelled sufficient blood to produce a palpable temporal pulse. About 2 cc. of 2 per cent procaine hydrochloride was injected into the blood through the right auricle. The heart was flabby. An electrical shock was sent through the heart about five times. This did not destroy the fibrillary movements. The heart was very flabby but of good color. Solution of calcium chloride was given through the right auricle. This improved the tone but fibrillation continued. I desired to increase tone and irritability, and, contrary to past experience, I gave some epinephrine. This increased the tone and I could feel the heart squirming in my hand. After a few moments the heart was beating. Fibrillation had dropped out and was replaced by a co-ordinated contraction at 11:05. Epinephrine hydrochloride was given in an intravenous drip. The incision was closed. An electrocardiogram showed auricular fibrillation, right bundle branch block and right axis deviation. The radial pulse was felt easily. There were no reflex responses. Mechanical respiration was carried out throughout. The pulse continued until 3:00 o'clock when it disappeared.

I should like to refer to Prevost and Battelli[5] for the introduction of the electric shock for defibrillation of the ventricles, to Wiggers[6] and to Hooker[7] for physiological studies in ventricular fibrillation and to Mautz[8] for the introduction of procaine to reduce the irritability of the heart. Reports in the literature on cardiac massage[9] are very numerous. The majority of these are from England and were in patients who were given chloroform.

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THE TREATMENT OF INJURIES TO THE CHEST*

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NEW YORK, NEW YORK

INTRODUCTION

IN the present war the casualties among the civilian populations are so widespread it is most timely that we discuss chest injuries with reference to their occurrence and management in modern warfare, as well as the treatment of them in everyday life.

At the December, 1940, meeting of the Royal Society of Medicine, Mr. Thomas in his discussion quoted Hoche's review of 11,000,000 English, French, American and German wounded:

DISTRIBUTION OF WOUNDS

	Per Cent
Limbs	68
Head, face, neck	15
Spine and cord	4
Pelvis	3
Thorax	6
Abdomen	4

MORTALITY IN ABOVE SERIES

	Per Cent
Total mortality	8
Wounds of abdomen	68
Wounds of thorax	56

WOUND DISTRIBUTION AMONG 12,350 KILLED

	Per Cent
Head	47
Thorax	20
Abdomen and pelvis	11.8
Limbs	9.9

The high immediate and late mortality of wounds of the thorax is evident from the above tables. Patients with injuries of the chest suffer from the same shock, hemorrhage and sepsis as those with wounds elsewhere, *plus* special factors which interfere with their respiratory and cardiorespiratory function and reserve. These are: (1) air and blood in one or both pleural cavities, frequently under increasing tension; (2) open pneumothorax with lung collapse, mediastinal flutter, paradoxical respiration with swing of the intrapulmonary air from one lung to the other and loss of the aspiratory effect on the great veins with decreased cardiac output; (3) cardiac tamponade, and (4) surgical emphysema—sometimes progressive. All of these features become accentuated in older people and in those with existing lung or heart pathology.

As mentioned in previous papers, since all injuries of the chest imply a potential pleural, lung or heart involvement the respiratory mechanism should be considered as consisting of three components: (1) The purely volumetric or spatial attributes of the thoracic cage, bronchial tree and lungs: their structure, vital capacity, tidal and reserve air, and the rôle they play in pulmonary ventilation; (2) the intrapulmonary function of gas exchange, and (3) the very important cardiopulmonary relationship. The two primary principles, therefore, in treating these wounds are, *first* the immediate treatment of the shock and hemorrhage by the proper administration of fluids—whole blood, plasma or serum, and crystalloids—together with the use of 100 per cent oxygen, and, *second* the restoration and maintenance of the normal anatomy and physiology of the cardiorespiratory mechanism as soon as this is possible. Depending upon the individual case and the conditions presenting as in active hemorrhage, extensive wounds of the thoracic wall and wounds of the heart and pericardium, the treatment of shock and hemorrhage and of the injury itself are carried out simultaneously.

GENERAL TREATMENT OF INJURIES

Before taking up specific types of wounds certain basic factors of treatment should be understood. First, almost all patients with

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wounds of the thorax suffer from shock and some also from hemorrhage. If there is a sucking wound present, this should be promptly sealed with a sterile pad strapped snugly in place, or, if this is not available, with anything at hand. Hemorrhage from intercostal vessels or the parietes may be temporarily controlled by a mushroom type of pack which is inserted into the wound and pulled outward, thus exerting pressure from the inside. The Shaeffer method of artificial respiration should be used with care as the manipulation may cause further injury to the chest wall or lungs. The legs should be elevated to combat shock and the patient transported in the Trendelenburg position unless it is evident that this causes respiratory embarrassment, in which case the head of the stretcher should be slightly elevated. The patient should be placed in bed with the head slightly elevated and as soon as possible a transfusion of whole blood or plasma given and 100 per cent oxygen administered. This may be done by the use of a B L B (Boothby, Lovelace, Bulbulian) type of mask, an oxygen tent if available, or failing these, nasal oxygen by catheter, or if necessary by attaching an anaesthesia bag over the canister of a gas mask or respirator and connecting this with an oxygen tank, as suggested by Marriott. Four or five liters a minute may be comfortably given by nasal catheter and about eight or nine liters with the B L B mask or the transformed respirator. Of these the catheter method is least effective. Morphine should be used as needed to allay pain and fear. It should not be given in such dosage as will depress respiration or productive cough, however, for the maintenance of good pulmonary ventilation and the ability to cough up secretions is essential. In the case of rib fractures, particularly when they are multiple, injections of 2 to 5 per cent novocaine into the intercostal nerves paravertebrally or at the site of fracture are most effective and may be repeated as necessary.

Almost coincident with the above general measures an accurate history should be obtained, postero-anterior and lateral *x*-rays of the chest taken in as near an upright position as possible and a careful physical examination made with certain rather general findings in mind. Any asymmetry, deformity, fulness or unequal motions of the chest should be noted. Also the position of the trachea, the apex of the heart and cardiac dulness and the level of dulness from fluid in the chest should be ascertained. Auscultation is important especially with reference to the heart and the possibility of cardiac tamponade, and likewise for the detection of intestinal sounds in the chest, as well as the signs of fluid or air in the pleural cavity. Blood pressure determinations should of course be obtained. Not infrequently the abdomen is held rigidly in wounds of the lower chest and here the decision must be made as to whether or not there is accompanying intra-abdominal, renal or spinal injury, sometimes an extremely difficult differentiation.

WOUNDS REQUIRING IMMEDIATE OPERATION

There are certain indications for immediate operative intervention, the most urgent of which is a wound of the heart with acute cardiac compression. This is first suggested by wounds close to the sternum and the diagnosis is made by (1) a falling or absent blood pressure, (2) distended veins of the neck with increased venous pressure, and (3) a silent heart by auscultation and by fluoroscopy. The patient also presents a rather grayish cyanosis. Prompt intervention is urgent with repair of the cardiac wound and thorough irrigation with warm saline, a transfusion of blood or plasma and oxygen therapy. Opening of the pleura is to be avoided and the wound is carefully closed. Aspiration of the pericardium may be tried as a temporary expedient to relieve the tamponade although one must remember that such relief of tamponade may permit active bleeding again from the wound in the heart. The sulfonamides should not be used directly in the pericardial cavity because of the marked fluid

response they provoke when employed locally in serous cavities or in the tissues.

Progressing hemorrhage requires operation. Generally, this is due to injured intercostal or internal mammary vessels, less often to trauma of the lung itself. Lung wounds, after the initial loss of blood, usually seal themselves because of the resulting collapse of the lung and the pneumohemothorax which ensue. Occasionally, however, if one of the larger vessels is damaged, open thoracotomy and suture or resection of the affected lobe may be required. Wounds of entrance and exit should be carefully explored so that the intercostal vessels may be carefully inspected as these are the usual sources of persisting hemorrhage. Foreign bodies and fragments of bone should be completely removed, the rib ends left smooth, the wounds débrided and the chest closed. Closed intercostal drainage should be established with suction up to 12 cm. of water if possible. If available, sterile sulfanilamide or sulfathiazole powder may be left in the pleural cavity and soft parts in 6, 8 or 10 Gm. amounts as prophylaxis against infection.

Sucking wounds of the chest demand immediate operation. The same meticulous wound toilet should be performed as just described. If the defect cannot be entirely closed, the muscles and skin should be drawn together as well as possible, the lung sutured snugly into the remaining opening and dependent closed drainage established (Barrett). A sterile vaseline gauze dressing is applied over the wound and strapped firmly in place so as to provide an aseptic, airtight covering.

Compound fractures of the ribs and wounds of the soft parts of any magnitude require débridement and closure if seen within the first eight hours. If there is any question about the wisdom of primary closure, the muscles may be sutured and the skin left open about a sterile vaseline gauze pack and later a secondary suture used if necessary. If large or grossly contaminated foreign bodies are present in the pleural cavity or lungs, they should be removed as soon as practicable as they are prone to cause hemorrhage and infection. If an injury to the diaphragm is suspected or if one is found in the course of wound exploration for one of the above reasons, the wound in the diaphragm should first be enlarged and the upper abdominal viscera examined for injury; only after this has been done should the diaphragmatic rent be repaired. Depending upon the amount of intraperitoneal damage it may or may not be necessary to open the abdomen as well as the chest.

In the convalescence from these procedures reliance is placed first on ample oxygen therapy and transfusions, then on a full diet rich in vitamins and on the sulfonamides for pneumonia or infection. A free airway and lung ventilation is of the utmost importance in the prevention of atelectasis and pneumonia. If the patient is unable to get rid of accumulated secretions, these should be aspirated as often as required by a catheter passed through the nose into the trachea and suction applied. This simple procedure may eliminate the need for bronchoscopy or may be used to supplement it. Fluid collections must be looked for and removed and the drainage tube kept open. Every effort should be made to facilitate early re-expansion of the lung and restoration of the individual to normal. Fluid accumulations, sepsis, pneumonia and secondary hemorrhages are the most common complications. In the absence of infection we have found suction up to 12 or 15 cm. of water an aid in re-expansion of the lung. If this is not available, the cavity may be washed with oxygen and the drainage tube then clamped for twelve to twenty-four hours and the process repeated. As the oxygen is absorbed an increasing negative pressure develops with resultant pull on the lung. If the lung expands well and there is but a small amount of serous drainage, the tube may be removed in a week. If sepsis develops, however, wide open drainage is established as soon as walling off has occurred sufficient to stabi-

lize the mediastinum. The cavity may be packed for a few days or suction may again be established. This helps keep the dressings dry, and, with dressings closely applied about the tube, will maintain an increasing degree of differential pressure. The presence of a bronchial fistula in no wise contraindicates the employment of suction. Later in the convalescence, physiotherapy and breathing and postural exercises are exceedingly helpful in restoration of the mobility of the thorax and expansibility and expansion of the lung, thus preventing fibrosis of both lung and pleura, scoliosis and deformity of the thoracic cage.

HEMOTHORAX

According to Edwards and Davies about 70 per cent of all chest wounds are complicated by hemothorax. This was the topic of an entire evening's discussion by the Royal Society in December, 1940. All agreed that when it is present with wounds like those just described débridement and thoracotomy is the indicated treatment. Frequently it occurs following crushing injuries of the chest with simple rib fractures and almost always it accompanies penetrating gunshot wounds and then may be called a "compound" hemothorax. In civil life these penetrating wounds are commonly made by small objects such as ice-picks, small knives or bullets; in war they usually result from bullets and shell fragments. A compound hemothorax with small clean wounds of entrance and exit as from rifle or machine gun bullets and small penetrating shrapnel fragments or stab wounds, should be treated like a simple closed pneumothorax. We have preached this for many years and I have referred to it in two previous articles. The important principle of therapy is aspiration with air replacement. Weeks, in his work with the New York police force, corroborates this, for he found that seventy-six gunshot wounds of the chest, treated without or with late aspiration, had a 26 per cent mortality, whereas in seventeen treated with early aspiration and air replacement there was no mortality, complete lung re-expansion in all and early return to duty. The unanimous opinion of this meeting of the Royal Society was in favor of this method of handling these cases and a similar opinion was recently expressed by Bumm, in Germany. When blood is present in the pleural cavity, an immediate inflammatory reaction of the pleura is produced with congestion and edema, copious fluid secretion and deposition in fibrin on both the parietal and visceral surfaces. This collection of blood and fluid increases the danger of infection, atelectasis and pneumonia. It absorbs slowly and unevenly with frequent loculations and sometimes the lung never completely re-expands. The result is an incapacitating fibrothorax, occasionally with deposition of calcium, with deformity of the chest and marked dysfunction of the affected lung from fibrosis, compensatory emphysema of the opposite lung and displacement of the mediastinal structures to the affected side. Thus the spatial and cardiorespiratory components of cardiopulmonary function may be severely damaged and such patients become permanently incapacitated. (Fig. 1.)

After the initial treatment of the shock and hemorrhage, dyspnea and cyanosis due to tension pneumothorax or fluid accumulation with mediastinal displacement must be relieved by repeated aspirations or the establishment of closed drainage. Roentgenograms should be made in the upright position and in interpreting these it is well to remember that the diaphragm on the affected side is usually elevated and that blood clot and air occasionally give a picture resembling coils of intestines. Livingstone suggests giving a glass of water with 4 Gm. of sodium bicarbonate before the *x*-ray to outline the diaphragm more clearly when the lesion is on the left. Barring emergency aspiration the chest should be aspirated in from twelve to forty-eight hours, and *all* the fluid removed and replaced with an equal amount of air. Such aspiration is threefold in purpose: (1) It is

an important diagnostic aid; (2) it prevents infection, and (3) it insures an early return of normal function. This should be repeated as often as fluid reaccumulates in sufficient amounts to cover the diaphragm. One tube of fluid removed should be sent to the laboratory for direct smears and cultures and a second kept at the bedside for future comparison. Furthermore, unless this fluid is removed collapse of the lower lobe and even large foreign bodies may be quite hidden by the fluid shadow. These aspirations are best performed with a large needle (14 to 16 gauge) attached to a two-way stopcock on the syringe so that as the fluid is removed the syringe need not be disconnected as it is filled with the air that is to be put back into the chest. Open thoracotomy is indicated if a foreign body of appreciable size is present, if infection develops or if it is noted that the clot cannot be removed and the pleura is becoming thick so that the lung fails to re-expand. No permanent pneumothorax should be left

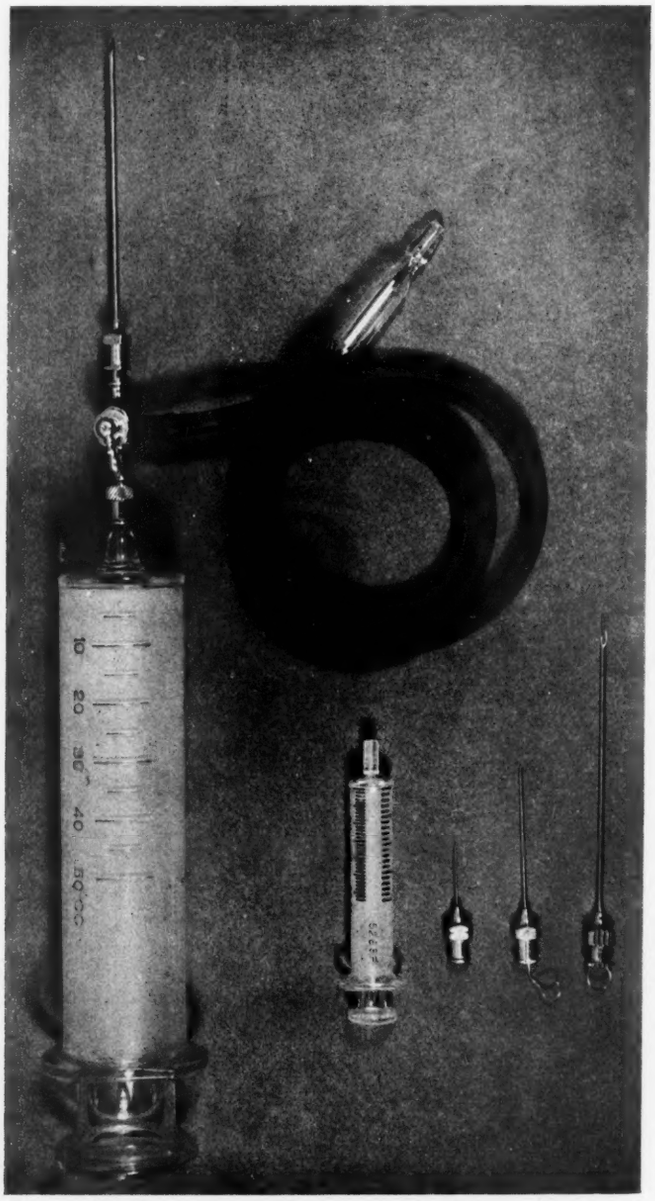

Fig. 1. Simple apparatus that may be used for aspiration of hemothorax and replacement of air.

—sterile or infected—for this is a source of future trouble and loss of function. The lung and chest wall *must* be brought into complete apposition by full expansion of the lung if possible. If this fails, a decortication or collapsing operation on the chest wall should be performed. An unexpanded pneumothorax is a potential source of trouble and should never be allowed to remain.

NONPENETRATING WOUNDS

Nonpenetrating wounds of the chest may vary in extent from slight contusion of the chest wall to extensive crushing with multiple rib fractures and tear of the underlying lungs. Such injuries are common among persons buried in débris, in aviation accidents and in sudden crushing injuries as when the victim is caught between a truck and a wall. Even simple contusion may cause an accumulation of clear fluid or an underlying atelectasis or pneumonia. Such sequelae are more likely in the aged or in those with existing pulmonary pathology. If ribs are fractured and the lung torn, a traumatic tension pneumothorax may develop rapidly and demand prompt relief. Frequently a hemothorax appears which should be evacuated and treated as already outlined. Subcutaneous emphysema is commonly present and usually absorbs spontaneously; if, however, it becomes so extensive in the neck as to endanger respiration, incisions should be made in the lower neck and the carotid sheaths opened.

Flying objects with violent impact on the chest wall may cause serious hemorrhage and laceration in the lung directly beneath the blow. Following a severe crushing of the chest there may be multiple rib fractures, which Barrett calls the "stove-in" type. These injuries may be complicated by a traumatic asphyxia or an acute dilatation of the stomach with inability to get rid of the vomitus, which in turn favors the development of pneumonia and pulmonary suppuration. In this compression asphyxia the lesions may be symmetrical in both lungs with generalized congestion, some edema and small subpleural hemorrhage and sometimes emphysema along the lines of the ribs; or there may be laceration or fracture of the trachea, bronchi or lungs.

I have recently had under my care a patient who sustained a fracture of his right main bronchus following a severe crush of his chest five years previously. This resulted in an almost complete atresia of the bronchus with repeated attacks of acute suppurative pneumonitis. The old fracture was demonstrated at operation. A subtotal pneumonectomy and later a thoracoplasty were performed with recovery. In all such injuries adequate pulmonary ventilation is best maintained by strapping the affected side to allay pain and control paradoxical respiration, injecting the intercostal nerves with novocaine to promote free respiration, and oxygen therapy of 60 per cent or more. Intra-abdominal or retroperitoneal lesions should always be looked for and may be extremely difficult to exclude because a rigid abdomen is common to all. Also, blunt cardiac trauma should always be suspected in these injuries and careful note made of pain distribution, blood pressure, quality of heart sounds, rhythm and murmurs, and electrocardiograms should be made at frequent intervals if possible. If evidence of heart injury is found, absolute rest and oxygen therapy should be instituted just as in the treatment of coronary thrombosis.

BLAST INJURIES

In the English literature considerable attention has been given to the blast injury or concussion and cases have been reported in detail by Dean and Thomas, Falla, Fallon, and Hadfield and Christie. The first experimental work was reported in 1924 by Hooker, who said bruising and rupture of the lungs "were the single gross lesions found postmortem after exposure to air concussion due to gun-blast or high explosive." Blast produces a sudden marked fall of arterial and venous pressures and a later washing out of venous carbon dioxide. Since the onset of the present war

further studies have been made under the direction of the Home Office by Barcroft with goats, and by Zuckerman with mice, pigeons, guinea pigs, rabbits and monkeys.

Zuckerman's work is of sufficient interest to report in detail. He used a seventy pound charge of high explosive and first explains the physics of a blast. Blast consists of two waves, a compression wave that lasts 0.006 seconds and a suction wave of 0.03 seconds. All pressures are, of course, in excess of the normal atmospheric pressure of fifteen pounds per square inch. The blast wave consists of a shell of compressed gas increasing very rapidly in radius—1500 feet a second at thirty feet for a sixty pound charge. There is an additional wind pressure exerted by this shell of gas *only* in the direction of its motion; hence any surface not directly in its path is subject just to the compression wave. This wave of pressure is highest in the region of the explosion and falls rapidly. At fifteen feet from a 125 pound charge the compression wave carries a pressure of 200 pounds. Close to the explosion the wind pressure may be as great as the pressure of the charge itself but it falls more quickly with distance. Thus everything in the immediate neighborhood of a big bomb is suddenly exposed to a wave of many times the atmospheric pressure, whereas at fifty feet this will be only two or three atmospheres and at 100 feet only a fraction of an atmosphere. The velocity and duration of the pressure wave is such that a human body will be completely immersed in it. The suction wave is much weaker and can never exceed fifteen pounds per square inch, which is a perfect vacuum. The magnitude of the pressure and suction vary directly with the amount of explosive. If an object is not blown apart by the wind and compression waves, it may then be attracted to the center of the explosion by the weaker and longer suction wave.

If the victim of a bomb explosion is not blown apart he may be injured in three ways: (1) by being hit by masonry, fragments or flying objects, (2) by being violently thrown, or (3) by the effect of the blast wave without being thrown. This last is the true blast injury or concussion in which there is no sign of any external injury, and even in this group Roberts warns of confusion with carbon monoxide poisoning.

In his work with animals with the seventy pound blast Zuckerman found that no animals died that were more than eighteen feet from the explosion and none more than fifty feet distant was injured. Furthermore, there was no sign of external injury in any of the animals examined. The outstanding pathological condition consisted of hemorrhages in both lungs and in all lethal cases blood was present in the bronchial tree and air passages. The blood came from torn alveolar capillaries as a rule. There was bruising or tearing of the lungs in the lines of the ribs, this bruising extending for a short distance into the parenchyma. Zuckerman concluded that these pulmonary lesions are caused by the pressure component of the blast by its impact with the body wall. This conclusion was based on the type of lesions found and further on the fact that animals encased in thick sponge rubber suffered little damage from the blast compared with their controls; also animals with one-half of their bodies protected by rubber sustained these lesions only on their unprotected sides when they were exposed with this side facing the blast and not on the protected side when this was the side exposed to the blast.

In the clinical cases similar findings have been recorded. After blast concussion symptoms may be delayed in appearance up to two to five days and consist of tachycardia, restlessness, dyspnea and cough with blood-tinged sputum. The signs are a fulness of the lower chest with diminished movement of the dipahragm, some spasticity of the upper abdomen, dulness and râles at the base of the lungs and a loss of translucency by *x*-ray with evidence of diminished rib expansion. The treatment is absolute rest, sedation and oxygen therapy.

TRANSPORTATION

In all of these patients with chest injury the possible risks of transportation should be recognized and weighed. The decision must be made with due regard to the personnel and facilities for caring for them. Emergency therapy includes the initial measures for shock and hemorrhage and then an appraisal of the problem at hand as to whether immediate or early surgery is indicated, whether hemothorax should be relieved with air replacement or observation and expectant treatment should be instituted. Here the question of transport enters. It is evident that these patients will have a better prognosis and fewer complications if they can be treated in institutions or centers familiar with and equipped for the care of thoracic surgery and trauma. In war organization such centers can be formed both for the reception of such injuries and for consultation and instruction. In civil life, for the more complicated patients, well equipped hospitals should be utilized and the aid of those interested in thoracic surgery sought.

ANESTHESIA

In all surgery of the chest anesthesia plays a most important rôle; this is particularly true in wounds in which it may be imperative to carry out a major procedure upon a person suffering from hemorrhage and in shock. Induction must be quiet, abundant oxygen must be supplied, effective respiration must be maintained with a free airway and bronchial secretions must be promptly removed. The anesthetist should, in addition, assume the management of the patient during the operation advising as to the extent of the procedure and the need of blood and other fluid. A quiet induction is best assured first, by proper sedation with morphine and scopolamine and perhaps a barbiturate given one-half hour previously. Intravenous pentothal sodium is also an excellent method of rapid and quiet induction. The agent itself may be selected from ether, chloroform, cyclopropane and ethylene. Of these ether is the safest and best. Both ether and cyclopropane can be used with a high percentage of oxygen but ether is less depressing, less liable to accident, is safer except with a person thoroughly familiar with cyclopropane, and, if properly administered, should produce little if any more irritation. In war and for mobile units ether and chloroform have the further advantage of greater adaptability for supply, storage and transportation. The method of *controlled* respiration with carbon dioxide absorber technic is strongly recommended (Nosworthy). An intratracheal tube inserted under direct vision, or as a Magill tube through the nose, is advised but is not essential as in these cases secretions are usually not a problem; hence an ordinary intrapharyngeal airway will often suffice. Intratracheal anesthesia, however, should be employed when possible, and in any event for all but emergency procedures. This assures better control of respiration and a free airway with the ability to aspirate secretions completely. There should be an airtight fit between mask and face so that the control of respiration may be taken over entirely by the anesthetist. This permits normal physiological respiration to occur even with the chest wide open and completely abolishes paradoxical respiration and mediastinal flutter. It also insures a plentiful supply of oxygen, the removal of carbon dioxide and quiet respiration. Hyperventilation hinders the work of the surgeon and may be harmful both locally to the wounded lung and generally in the washing out of carbon dioxide and accentuation of shock. Controlled respiration likewise allows occasional inflation of the lung during operation and re-expansion of it at the completion of the procedure. An elaborate apparatus is not required for this anesthesia technic and while an absorber is helpful it is not essential. All that is really necessary is an airtight mask with the bag kept not quite fully distended; this will then permit of control of the respiration by rhythmic hand

pressure on the bag. Oxygen therapy should be maintained for at least twenty-four hours postoperatively. It might be pointed out here that many restless and apparently delirious patients will frequently respond most dramatically to oxygen therapy alone for serious anoxia may exist without any cyanosis or dyspnea, particularly in the presence of anemia.

SUMMARY AND CONCLUSIONS

Wounds of the chest and their treatment have been discussed. Almost all are accompanied by some shock. This and hemorrhage when present should be treated before anything else is attempted.

Immediate operative intervention is indicated for wounds of the heart and pericardium, progressing hemorrhage and sucking wounds of the chest. Early operation is also indicated in compound rib fractures, moderate and large wounds of the soft parts, large or infected foreign bodies and possibly wounds of the diaphragm. Simple perforating wounds, unless accompanied by hemorrhage from an intercostal or internal mammary vessel should not be operated upon. Tension pneumothorax should be promptly relieved and subsequently fluid and air removed as indicated.

Traumatic hemothorax should be treated by aspiration and air replacement. Expectant treatment is to be condemned. Operation may be required *later* for sepsis, foreign body or organizing blood clot.

Crushing and nonpenetrating wounds of the chest are discussed. The treatment is primarily conservative with strapping, sedation and novocaine injection of the intercostal nerves. Severe lung or bronchial injuries should be suspected and may require surgery later. Contusion of the heart, rupture of the diaphragm and intra-abdominal injury should likewise be borne in mind.

Blast injury or concussion is considered in detail. The injury is probably a direct contusion of the lungs from the compression wave of the blast, similar to a very severe blow on the chest.

The problem of anesthesia is discussed and an ether-high oxygen mixture administered by the closed system technic with controlled respiration and intratracheal intubation recommended.

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RECENT EXPERIENCE WITH WAR WOUNDS OF THE CHEST

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DURING the latter part of the last war, much interest became focused on wounds of the chest owing to the new conception that thoracic injuries should be treated on the same principles as wounds elsewhere in the body. The conclusions reached as a result of this fresh outlook have been fully described (Duval,[1] Grégoire and Courcoux,[2] Gask,[3] etc.).

During the subsequent years of peace, steady progress was made in thoracic surgery. It is, therefore, important that we consider in what way this advance has influenced the treatment of chest casualties since the disastrous outbreak of further hostilities. The prelude to the present vast conflagration occurred in Spain and those responsible for the care of chest wounds in the war of 1939 naturally sought the experiences of the surgeons who treated the Spanish casualties. Jolly[4] leaves the impression that few primary intrathoracic operations were performed for he states that "the outlook for all cases requiring an intrathoracic operation is grave" and "a lung abscess developing later in a living man is to be preferred to an aseptic thoracic cavity in a dead one." In general, Jolly recommends the conservative treatment in vogue during the early phases of the 1914 war. It appears, therefore, that no advance in the care of thoracic wounds evolved from the Spanish Civil War.

Although a considerable amount has been written on chest wounds since the outbreak of the present War, most of the opinions expressed are based on experiences of over twenty years ago (Tudor Edwards,[5] etc.). This is partly due to the fact that military casualties have been mercifully few and that chest wounds in civilians resulting from aerial bombardment tend to be severe and often result in death before reaching the hospital. Compared, therefore, with the almost astronomic number of casualties dealt with in the Great War, the experience derived during the last two years has been very limited. However, it is not proposed in this communication to re-state the generally recognised features of war wounds of the chest but to consider any new factors which have arisen and to what extent the last two years have modified or confirmed the 1920 conceptions.

Personnel. If Duval's results with thoracic wounds are compared with those obtained in the latter part of the last war from large sections of the British Army (figures quoted by Gask[3]), it seems at first doubtful if the general adoption of Duval's policy of frequent early surgical intervention in penetrating wounds was justified.

There are many factors which influence individual group results, in particular the nature of the missiles being used and the condition of the soil and men's clothes. But, after adequate allowance has been made for these factors, there remains a discrepancy in results which is at first confusing. This discrepancy is readily explained by the fact that there are very large numbers of general surgeons with excellent training but less than 5 per cent are accustomed to thoracic work. It would be futile to pass comment on this fact if it did not have a direct practical bearing on the treatment of chest injuries. Apart from the obvious conclusion that war calls for a higher percentage of thoracic surgeons and that those existent should be organized into readily available teams, it must also be clear that the treatment of many individual cases must depend upon the thoracic training of the surgeon and, in addition, upon the

facilities for the after-care of such cases. A patient with a penetrating wound may have better prospects of recovery with conservative treatment if those conversant with this region are not available.

Transportation. It is usually stated that the general condition of patients suffering from thoracic injury suffers very severely from transportation. When the wounded, untreated except for the application of dressings, reached England during the evacuation of France, it was thought that patients with severe chest wounds would be unlikely to survive the journey; yet there were a number who did and the general condition of many was not too bad. When the bombing of London started, chest casualties were admitted to many hospitals scattered over the Metropolitan area. A decision had to be made in each case whether the best chance would be given to the patient by operation at the hospital of admission (where he would have to be retained for several days after operation) or by transfer to one of the chest units stationed in hospitals some twenty miles outside the center of the city after adequate resuscitation. As time passed, it became a routine (with few exceptions) to advocate retention at the primary hospital for those who did not require early open operation, e.g., crushing injuries, and the transfer of those who did, as soon as morphia, warmth, blood transfusion, etc., had overcome the initial shock. The only local measures taken prior to transfer were prevention of sucking in open pneumothorax cases and the control of obvious external hemorrhage. The patients suffered very little from the journey. In two cases—a girl of twelve with a thoraco-abdominal wound involving the liver from which she was still bleeding, and a boy of seventeen with the internal mammary artery severed and bleeding into the pleural cavity—it would have been preferable to continue the transfusion during the journey; but, in spite of not doing so, neither patient deteriorated except for a slight further rise of pulse rate. Patients are distressed by movement in bed or from bed to stretcher and vice versa; but, if this is carefully done and the patient made comfortable on the stretcher with the trunk raised to 30 degrees to the horizontal, the journey itself is well tolerated provided the driver realizes the nature of his cargo.

We would, therefore, recommend the transportation of patients requiring primary operation to a hospital where there are proper facilities for treating them, expecially as postoperative care is frequently more important than the operation.

"Blast" or Pulmonary Concussion. During the 1914–1918 war, it was noted on many occasions that men might be found dead in the immediate vicinity of an explosion without external evidence of injury. No satisfactory explanation of this phenomenon was provided although massive pulmonary collapse (Lockwood[6]) and acute carbon-monoxide poisoning were suggested as possible causes. At the onset of this war, Phillips[7] stated that he had performed many postmortem examinations on miners found dead as a result of explosions in the South African gold mines and discovered multiple scattered intrapulmonary hemorrhages. Zuckerman[8,9] then published the results of his experimental work on animals showing that the positive pressure wave of an explosion, acting directly on the chest and abdominal walls, may produce extensive diffuse intrapulmonary hemorrhage. This was found to be the most consistent autopsy finding in animals dead as a result to exposure to "blast." Hadfield[10] and his co-workers later confirmed these findings in man from their examination of victims of aerial bombardment.

Clinically, very few patients have been recognized as suffering from this condition. It would appear that, in man, there is only a very small margin between immediate death and complete escape from "blast" injury. Alternatively, the cases have not been recognized owing to ignorance of the symptoms and signs but this is improbable as there are many observers who have been only too ready to make the diagnosis.

At present, it can only be stated that pulmonary concussion should be suspected in any patient found close to the site of an explosion, particularly if there is cyanosis, marked shock, dyspnea, diffuse chest or abdominal pain, scattered pulmonary râles or abdominal rigidity unaccounted for by any other injury. From the surgical standpoint, two observations are worthy of stress: First, generalized abdominal rigidity of such degree as to precipitate exploratory laparotomy has been observed by O'Reilly[11] and others; this is probably accounted for by associated hemorrhage into the intercostal spaces which causes irritation of the intercostal nerves. Second, there is evidence that the hemorrhage continues for some time after the injury. Inhalation anesthesia, which might otherwise be required for treatment of co-existent injuries, should therefore be avoided if possible.

Closed Injuries. Since the aerial bombardment of the civilian population started, the proportion of closed to penetrating injuries has steadily increased, presumably as a result of the population taking more adequate protection. Damage to the viscera with the thorax without fracture of the bony framework is well recognized, particularly in children owing to the elasticity of their chest wall. We have seen three cases of closed traumatic pneumothorax and three cases of gross pulmonary contusion in the absence of fractured ribs. Similarly, visceral damage may occur when a missile penetrates the soft tissues overlying the thoracic skeleton. For this reason, hemoptysis is not always evidence of penetration of the chest wall.

As regards the diagnosis and treatment of closed fractures of the thorax, there is little to add to the generally accepted views. Provided adhesive strapping used to splint fractured ribs extends at least 3 inches *beyond* the midline anteriorly and posteriorly, there appears to be no necessity for encircling the entire chest which has the disadvantage of diminishing the function of the opposite lung. We have found this to be true with fracture of as many as eleven ribs on one side and with severe degrees of "stove-in" chest where part of the chest wall moves paradoxically. Theoretically, inelastic strapping provides the best splint but, in practice, elastoplast or its equivalent can be put on more evenly, remains in the applied position longer and can be made to give equal support if slightly stretched. Strapping should be postponed if the patient is severely shocked as the movement entailed in its application may result in dangerous exacerbation of the circulatory depression. During the period of recovery from shock, the patient should be rolled slightly towards the affected side so that respiratory movement is limited on this side and any paradoxical movement is minimal unless situated anteriorly.

Rib fracture with gross displacement of the fragments may be associated with damage to the intercostal vessels giving rise to progressive intrapleural hemorrhage which may necessitate surgical exploration. Serious damage to an upper abdominal viscus may indicate laparotomy. With these two exceptions, it is probable that closed fractures of the ribs even when there is gross comminution and displacement of the fragments, are best treated conservatively, i.e., simply by strapping. We have treated all such cases, including those with evidence of pulmonary damage, in this way and have been surprised at the excellent functional end results. If the patient develops a hemothorax which is not progressive, the blood should be aspirated (with air replacement if necessary). The escape of air from the lung into the pleural cavity may be ignored unless giving rise to respiratory distress. In the latter case, the pneumothorax tension is most easily relieved by inserting a short wide-bore needle into the pleural cavity and connecting it by rubber tubing to a "water-seal" drainage bottle.

Brock[12] has drawn attention to the occasional occurrence of acute dilatation of the stomach in crushing injuries to the chest. Even if there is no vomiting, this complication should be considered when the pa-

tient's general condition is much worse than can otherwise be accounted for. If the lower part of the left side of the chest is the site of injury, delayed rupture of the splenic capsule is a possible contingency. One of our patients developed massive intraperitoneal hemorrhage on the ninth day.

Diaphragmatic herniation should undoubtedly await surgical cure until the patient has otherwise made a complete convalescence.

Sternal fractures may occur apart from those resulting from direct trauma. We were forcibly reminded of this when a convoy of wounded men was received after escaping over the cliffs at St. Valery in France. The chief complaint of three of the men was pain on breathing and they were accordingly allocated to the thoracic unit. After admission, examination revealed a transverse fracture of the sternum, in each case complicating crushing fractures of the vertebral bodies which had resulted from their jump from the cliff top.

Penetrating Wounds. In the absence of pleural penetration, there is no comment to make on the well established principles of treatment formulated twenty years ago.

Hemothorax. In patients who do not require primary operation except for excision of entry and exit wounds, e.g., through-and-through bullet wounds, penetration by very small fragments of shell or bomb casing, the treatment of a hemothorax has been revised during the last two years. In the past, it was customary to await absorption of the blood unless the bleeding was progressive or causing respiratory distress from pressure. The disadvantages of such expectant treatment are: (1) An excellent bacterial culture medium remains in the pleural cavity thus encouraging infection and, if it occurs, the consequent empyema is frequently very large. (2) Absorption of the blood is often slow and the deposition of fibrin on the pleural surfaces is sometimes progressive. This fibrin gradually becomes organized, hampers further pulmonary re-expansion and limits movement of the chest wall. In a few patients, actual calcification of the thickened pleura or residual effusion occurs.

It was argued in favor of this policy of non-interference that aspiration encouraged recurrence of the hemorrhage and that the damaged lung healed more readily if collapsed.

It is now a general routine to aspirate as much blood as possible about twenty-four hours after the injury (always before the fourth day). The fluid is removed as long as it can be readily withdrawn through a large-bore needle. If the patient complains of a tight feeling in the chest or if considerable force is required to aspirate the blood, air is introduced to relieve the increasing negative pressure. Provided excessively negative intrapleural pressures are avoided, it is improbable that removal of the blood will cause recurrence of the hemorrhage. It has not occurred in our experience. There is very little evidence to support the common thesis that an injured lung heals better if collapsed. There is one objection to partial replacement with air. The air rises to the upper part of the pleural cavity and temporarily prevents the apex of the lung adhering to the parietes. If infection occurs early, an almost total empyema must follow. It is therefore advisable to limit the use of partial air replacement to those cases in which it is not possible to withdraw all the blood without leaving an excessively negative pressure. Grégoire and Courcoux[2] state that, in the absence of infection or gross tissue damage, the blood of a hemothorax does not clot apart from the deposition of a thin fibrin network on the pleural surfaces; this has been our experience. Except for this phenomenon, aspiration would always be difficult and very incomplete.

Infected Hemothorax. The diagnosis of infection rests finally on bacteriological examination (including anaerobic culture) of samples of aspirated fluid. The treatment of infected cases is exactly similar to that of any acute empyema.

So far, the evidence favors a diminution in the incidence of infection and a decrease in the severity of toxemia when infection

does arise as a result of the prophylactic use of the sulfonamide drugs. We have had three cases infected with Clostridium welchii and all have survived. In reference to this latter type of infection, the radiological appearances are often characteristic. When the major portion of the blood has been removed by aspiration, subsequent infection of the residium by gas-forming organisms causes the deposition of fibrin which becomes vacuolated as a result of gas production within its meshes. In a radiogram the appearance is not unlike that of a polycystic lung.

Contralateral Pulmonary Collapse. The cause of this complication remains unproved. Penetrating chest wounds initially make expectoration very painful and it seems almost certain that bronchial obstruction by secretion, or rarely blood, is the explanation of the collapse. It is, therefore, probably justifiable to bronchoscope and aspirate such cases if seen within twenty-four hours of the onset of the collapse. Later than this, removal of secretion is most unlikely to be followed by immediate re-expansion of the collapsed area. Collapse of the whole or part of the contralateral lung was a very common finding (about 16 per cent) at Casualty Clearing Stations in the last war. We have had only one such case. Re-expansion was slow but complete and bronchography prior to discharge from the hospital showed that bronchiectasis had not complicated the collapse.

Indications for Primary Operation. Primary operation may be indicated to control progressive hemorrhage, convert an open into a closed pneumothorax or prevent sepsis. In addition, most suspected thoracoabdominal wounds require exploration in order to repair the diaphragm and any damaged abdominal viscus. The exploration should be carried out through the chest in the vast majority of cases in this latter group (Gordon-Taylor[13]).

For those inexperienced in the treatment of penetrating wounds, it is difficult to decide on which patients to operate when prevention of sepsis is the sole indication. Through-and-through wounds when bony fragments have not been carried into the pleural cavity or lung are unlikely to be followed by sepsis. Retained fragments of shell or bomb casing less than one-half inch in diameter which have entered the chest through an intercostal space infrequently introduce infection and rarely give rise to later trouble. In nine years' work at a military pensioners' hospital after the last war, one of the authors (J. E. H. R.) saw only eighty cases in which retained missiles were giving rise to symptoms. On the other hand large missiles often carry in rib or other bone splinters and foreign material such as clothing; suppuration is then a common sequel.

A great deal of judgment is required to decide which patients to select for primary operation. A surgeon inexperienced in surgery of the chest would be wise to err on the side of conservatism.

Technic of Primary Operations. There are two new practices which merit attention. The introduction of an intercostal self-retaining catheter of the Malacot type prior to the closure of the thoracotomy wound allows the escape of air and effusion and obviates the necessity of postoperative pleural aspiration. It should be in the lower part of the pleural cavity although not actually in the costophrenic sinus as, in the latter position, the diaphragm may rise sufficiently to obstruct the drainage. The site of drainage should never be through the thoracotomy wound or that produced by the missile. The tube is anchored to the chest wall by transfixion with a sterile safety pin which is fixed to the skin with adhesive strapping. It is then connected by rubber tubing to a "water-seal" drainage bottle. The catheter is removed at the end of forty-eight hours and the wound of insertion closed with a single suture inserted at the time of the original operation. Poudrage of the pleural cavity with 10 to 15 Gm. of sulfanilamide before closing the chest is a convenient way of obtaining an adequate early concentration of the drug in

the blood stream and may also give rise to an increased local concentration inhibiting bacterial growth. Much of the drug is, of course, lost through the drainage tube.

Anesthesia. Anesthesia which provides a comparatively small respiratory excursion and does not cause anoxemia or a fall in blood pressure is required. Our patients have been given cyclopropane which fulfills these essentials extremely well but a trained anesthetist is required for its administration. Nosworthy[14] has described with enthusiasm the value of "controlled respiration." It has been employed on our unit when operating on the diaphragm, the immobilization of which is technically a great help; but our experience is limited to such cases. It is difficult to appreciate its advantages when the thoracic wound does not involve the diaphragm. Under most circumstances a mixture of nitrous oxide, oxygen and ether would probably give the most satisfactory results.

Breathing Exercises. This subject is well worn but the absolute necessity of giving all patients with thoracic injuries breathing exercises, shoulder girdle movements and postural training may be emphasized once again. Treatment should start very early during the patient's convalescence; in the absence of infection, gentle exercises and passive movements may begin on the second or third day. Full function will rarely be restored unless such treatment is given. In the case of military patients this means that few patients will be returned to the A1 fitness category. The masseuse's time is more than rewarded by the results obtained for many of the patients leave the hospital with their vital capacity increased to a level above that present before the injury.

These disjointed remarks omitting reference to many major sections of thoracic injury, e.g., wounds of the heart and pericardium, have been prepared in the hope that they may be of some value to those who are anxious to know whether the last two years of warfare have modified the treatment of chest wounds.

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THE PROBLEM OF WOUND SEPSIS IN THORACIC SURGERY*

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MANY of the wounds in thoracic surgery are susceptible to infection because of conditions which are peculiar to such operations. These may be listed as follows:

1. Frequently, the patients are suffering from a chronic disease and some of them may have a lowered resistance to infection.

2. An adequate incision necessitates wide exposure of tissue which may have a low resistance to the organisms which invariably enter every wound. Since the air of every operating room not only may, but usually does have pathogenic bacteria sedimenting from it onto the wound and sterile supplies, the larger wound becomes the more highly contaminated from this source.

3. There is inevitable trauma which is greater than in clean cut incisions in soft parts. This, of course, diminishes the resistance of the tissues to the growth of the bacteria which gain entrance.

4. Hemostasis is unsatisfactory in many cases because of the size of the wound, the many small bleeding points, the relative inaccessibility of certain bleeders and the possibility of bleeding coming on following closure of a "dry wound" as a result of uncontrollable motion associated with respiratory movements.

5. The dead space left following many operations cannot be obliterated. This is particularly true after the first-stage thoracoplasty with apicolysis or after a pneumonectomy.

6. The wound cannot be put at rest, as is best for healing, because of the respiratory movement and continuous changes in the intrathoracic pressure in the presence of a mobilized chest wall.

The wounds with which we have to deal may be divided into the following groups:

1. *Traumatic Wounds.* (In which contamination exists when the patient comes under treatment.) In these wounds which enter the pleural cavity careful first aid is of the utmost importance. The small, clean-cut wound should be closed after careful cleansing. The ragged wound with extensive trauma to tissues, and the possibility of the presence of clothing, wadding or any foreign material other than smooth metal, must be carefully explored as soon as the condition of the patient will permit it. It should be thoroughly washed out with a nonirritating solution and all foreign material, blood clot and severely traumatized tissue should be removed. Bleeding must be carefully controlled, the least irritating sutures and ligatures should be used to close the wound without drainage and with the minimal trauma to tissue and least constriction of blood supply.

At the present stage of our knowledge of sulfonamides, a suitable one of these should be sprinkled in the wound to inhibit bacterial growth. At times it may be necessary to shift a flap of soft parts to close a large defect opening into the pleural cavity, but the pleurae must be closed by some method if the condition of the patient will permit it.

If infection occurs, the empyema should be treated as any other empyema and the chest wall wound should receive adequate drainage but with support to limit the separation and if possible avoid a sucking wound, as is done in the case of the wound following pneumonectomy, as described later in this paper.

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The low grade infection in a pleural cavity filled with organizing blood clot demands special consideration. In these there may be multiloculated pockets which make drainage unsatisfactory while the fibrin and organizing hematoma prevent expansion of the lung and apposition of the pleural surfaces. While our experience with these has been limited, it seems to us that where this condition exists the thorax should be opened sufficiently to give adequate exposure, all the fibrin and organizing hematoma should be evacuated and the resulting cavity should be treated by irrigation until there is a clean granulating wound which can then be closed by the suitable application of suction.

2. *Large Clean Primary Incisions.* (Such as first-stage thoracoplasties or thoracotomies.) In these clean wounds contamination from sources other than the air we feel have been largely brought under control in the well run operating room. The wound itself, left by a well trained surgeon in the best possible condition for healing, can handle the few bacteria that gain entrance, if the relatively high contamination from the air during periods of widespread respiratory tract infection is brought under control. Infections in these wounds should be rare and seldom severe.

3. *Reopened wounds* such as second and subsequent stage thoracoplasties are susceptible to infection from at least two sources not present in the primary incision:

(a) The first of these is from the few organisms which may have gained entrance to the preceding wound, found a harbor about catgut sutures or in a pocket of fluid in a dead space, multiplied there but not to the extent of causing sufficient local or general reaction to be diagnosed as an infection, and are present in far greater numbers to be distributed through the tissues freshly exposed at the subsequent operation.

(b) The second of these sources is from organisms normally present on the skin which may have grown along the skin sutures of the preceding stage to form clusters of organisms which may be distributed through the reopened wound. When the reaction about the sutures is sufficiently great we have the well known stitch abscess, but there must be a less extensive growth of organisms about many other sutures.

Whatever the explanation, when the air as a source of wound contamination is eliminated, the reopened wound is more susceptible to mild or moderate infection than the primary incision.[12] On the other hand, in our experience when the air as a source of contamination is not eliminated the reopened wound is less susceptible to severe infections than the primary incision. Our explanation of this is that the patient at the time of the second operation has developed some immunity as a reaction to the contaminants which gained access at the previous exposure.

In our experience infections in reopened wounds, protected from air contaminants, have been mild, localized and in the subcutaneous fat or a pocket of fluid in a dead space.[12]

4. Certain wounds are potentially infected from the nature of the disease and the operation. Examples are those made for a lobectomy or pneumonectomy. If every precaution is taken to limit soiling when the bronchus is divided and the stump of the bronchus is buried beneath tissue with an adequate blood supply to prevent it from reopening, many of the cases will not develop an infection. The pleural cavity may or may not be drained, depending on the judgment of the operator as to the probability of infection. If infection occurs in the undrained pleural cavity, it is easy to drain through a tube inserted by the trocar and cannula technic. Where there is a large dead space which must be collapsed, as following a pneumonectomy, it is important to insert the tube so as not to interfere with an extrapleural paravertebral thoracoplasty, which then can be performed in a sterile field. If there is no bronchial fistula, the cavity can be drained, irrigated and partially collapsed

by suction; or if there is a bronchial fistula, continuous aspiration can be applied through a tube inserted well anteriorly. In either case treatment can be carried on indefinitely without leakage of pus around the drainage tube. The advantages of collapsing the infected pleural space down to a small residual cavity by means of operations performed in an aseptic field are too great to need further comment. The small residual cavity can be further treated by closed drainage, open drainage or by unroofing as the individual case may demand.

5. Wounds grossly contaminated by highly pathogenic aerobic, anaerobic or mixtures of organisms as follow drainage of a lung abscess, demand special consideration, since the secondary dissecting infections in the cleavage planes of the chest may present a greater threat to the life of the patient than the original disease. At one time it was our opinion that drainage of every lung abscess should be delayed until the chest wall incision down to the pleura had been packed sufficiently long to allow sealing off of the cleavage planes and the development of granulations over the incised tissues. However, with the preoperative and postoperative use of arsenicals intravenously to inhibit the fusospirochetal organisms, with the wound edges protected from the more gross contamination at the time of and following operation, with the wound left wide open for drainage and aeration, and more recently with the aid of the sulfonamides, we can in most instances, in which the pleural cavity over the abscess has been obliterated by adhesions, drain these abscesses in one stage.

The problem of wound sepsis logically falls into two divisions. The first of these is prophylactic or the avoidance of infection, while the second is concerned with the treatment of the infection once it has been established. In the case of clean wounds we should develop a technic which will eliminate most of the latter division so that an infected wound will become a rarity. This statement is made without reservation since such a condition exists in the operating rooms of the Duke Hospital today and can be easily duplicated elsewhere.[12]

Every phase of surgical technic has its bearing on whether or not the wound will develop an infection. Theoretically, if every bacterium could be kept out of the wound at the time of and following operation, we could not have an infection. Under these conditions we still could have poor healing as a result of traumatized tissues, collections of blood or serum, irritating sutures, drains, packs, etc. However, if anyone operates using such technic, it is all the more imperative that the number of viable bacteria entering the wound be kept to the minimum. Since some bacteria enter every wound, a goal of complete asepsis is unattainable and this makes it all the more important that we leave the wound in the condition best for healing in the presence of the lowest contamination that can be obtained. The ideal wound should be made with minimal trauma and closed with an accurate reapproximation of normal structures by means of non-irritating sutures, which do not strangulate tissues or interfere with their blood supply. There should be no dead space, no bleeding into the wound or tissues and no drains or packs should be inserted. The tissues should be placed at complete immobility in such a position as is conducive to good blood supply and good blood and lymph drainage. Since each of these more or less ideal conditions is unattainable, we must strive for the nearest possible approach to them.

The patient should be kept in the best possible condition before, during and following operation. His disease should be brought under the maximum control by adequate hygienic measures; his general nutrition, vitamin, fluid, and salt balance should be maintained at the optimum level. While rest may be very important in the treatment of tuberculosis, complete bed rest with its resultant atrophy of muscles, including the myocardium, and resultant

circulatory instability may be poor preparation for an operation.

Following operation the patient should receive rest, but not to the extent of avoiding frequent changes of position to improve the circulation, and to allow drainage of fluids from the dependent parts, particularly in the lungs and in the operative region. The local wound should be immobilized, but not to the extent of embarrassing respirations by tight bandages or keeping the wound dependent at all times in order to let the weight of the patient compress and splint it.

Fig. 1. (No. 77533.) A tumor of the left main bronchus of four years' duration caused complete obstruction with multiple abscesses thoroughout the left lung. The lung was removed on January 2, 1937.

Too frequent dressing of the clean wound results in needless inconvenience, fatigue and pain to the patient, and opens the wound unnecessarily to the dangers of infection. On the other hand, particularly during hot weather, the dressings on the fresh wound may become saturated with serum or a mixture of serum and perspiration; and if these are not removed, maceration of the epidermis along the suture line may result, particularly if the patient is permitted to lie on the operative side. Dressings performed with reasonable frequency and washing of the skin with alcohol may diminish the skin contaminants, avoid the development of stitch abscesses and result in better healing.

Even though, as stated above, we cannot prevent the entrance of every bacterium into a wound we should set this as the goal toward which we strive and exercise every precaution which may aid in its attainment. Among these precautions are the following:

The usually accepted aseptic technic with the sterilization of all instruments and supplies, careful skin preparation, isolation of the wound from the skin as soon as the incision is made, the covering of the carefully prepared hands of the operating team with sterile gloves, etc., etc., should be scrupulously and intelligently followed. This means that "aseptic technic" is not to be carried out by rote because it is the accepted thing to do but that every procedure is performed with a definite purpose in mind. These are determined by cultural checks on each step of the operative procedure, the results of which must be familiar to every member of the operating team.

An additional warning should be given in regard to the sterility of the prepared skin of both the patient and the personnel. Scrubbing with soap and water does not render the surface of the skin free of bacteria, but when this is supplemented by adequate chemical treatment, including the use of 70 per cent alcohol on the hands, the surface of the skin when cultured is relatively free of viable bacteria. On the other hand, at the end of the operation, particularly in hot weather when perspiration enters the picture, many of the skin contaminants may be on the surface of the hands of the operating team or skin of the patient at the operative site. This makes it imperative that every precaution be taken to avoid puncturing the glove, and that nothing touching the skin be permitted to come in contact with the patient's wound. If hemostatic forceps are to be left on the wound, they should be protected from the underlying skin

with a sterile towel and the wound itself should be protected by a second towel which overlaps the skin edges and covers

Fig. 2. (No. 77533.) The cavity following removal of the lung shown in Figure 1 developed a mixed infection including anaerobic organisms. A drainage tube was inserted on January 9, 1937, and the cavity was treated by closed drainage, tidal irrigation and suction for thirty days. Note the diminution in the size of the cavity brought about by elevation of the diaphragm, shift of the mediastinum and partial collapse of the chest wall.

Fig. 3. (No. 77533.) A first-stage extrapleural paravertebral thoracoplasty with removal of parts of five ribs was performed on February 8, 1937, thirty-seven days after the pneumonectomy and thirty days after the institution of tidal irrigation and suction. In such patients a larger number of ribs can be removed without serious disturbance to the respiratory mechanism since the patient has already become adjusted to the use of only one lung and the mediastinum can be stabilized by suction within the cavity to pull the walls of the cavity into apposition.

the clamps. The clamps can then be removed at the end of the operation without contaminating the gloved hand, or ligatures can be applied without contaminating the ligature as it slides along the clamp. The knife used for the skin incision should of course be discarded as soon as it has divided through the contaminated glands in the skin, and this part of the wound should be covered. These covers should not be removed until the operation has proceeded to the closure of all the underlying layers of tissue. They should then be removed, all exposed skin treated with a suitable antiseptic and recovered up to the edge of the incision with sterile drapes before proceeding with the closure or otherwise touching the skin.

Contamination of the clean wound, and exposed sterile supplies by organisms given off from the noses and throats of the occupants of the operating room, transported by way of the air, and sedimenting on the sterile field, constitute in our opinion the greatest danger of infection in these large wounds in the well run operating room of today.[7] We have long made a useful gesture toward the control of this danger by the conventional mask worn over the mouth and, in more recent years, almost universally over the nose. The importance of this masking in cutting down massive contamination from rapidly sedimenting large droplets cannot be emphasized too strongly. On the other hand, the generally accepted mask should not give one a false sense of security since it will not prevent a rapidly

increasing contamination of the air with small droplets or droplet nuclei which are the bacteria left after the liquid has evaporated, as has been emphasized in recent publications by Wells. Sneezing, coughing and talking should be prohibited or reduced to the minimum. The potential danger from the presence of a nose and throat carrier of highly pathogenic and virulent bacteria in the operating room is in our opinion immeasurably greater than is generally accepted by most surgeons today. Elimination of every transient carrier is difficult but the persistent carrier of highly pathogenic beta hemolytic streptococci or hemolytic yellow staphylococci should not be allowed in the operating room.

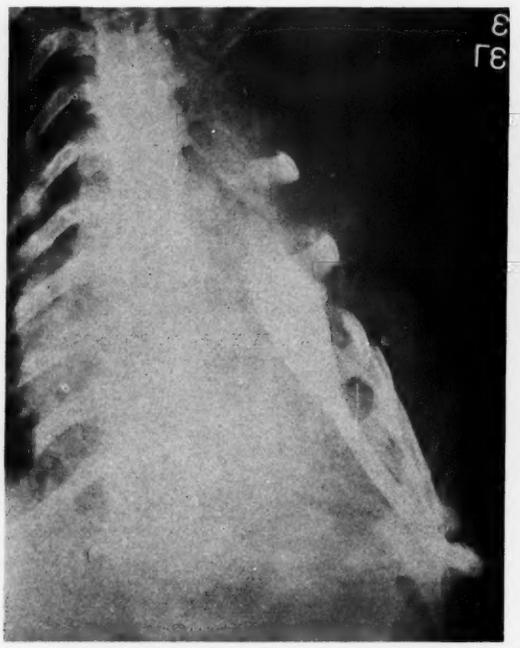

Fig. 4. (No. 77533.) On February 23, 1937, fifteen days after the first-stage thoracoplasty, the remaining six ribs were resected and the empyema cavity was treated by suction and irrigation for the next forty-two days. On April 6, 1937, the small residual cavity demonstrated here by lipiodol injection was collapsed by removing the ribs and thickened pleura overlying it and the soft parts were allowed to fall in. The patient was discharged with the wound almost healed sixteen days following the latter operation.

Air contamination can be kept down by limiting the number of occupants during, and the duration of occupancy before, these operations. It can also be reduced or held down by an efficient ventilating system which supplies large quantities of clean air (free of pathogenic bacteria) to the operating room.[2] The reserve sterile supplies can be protected partially by a mechanical sterile canopy which may reduce the number of colonies of viable sedimenting bacteria reaching the supplies by 50 or more per cent or by a canopy of bactericidal radiation which will kill all bacteria falling in an exposed position within one to two minutes. The two can be combined by placing the radiation unit beneath the mechanical canopy. The mechanical canopy is impractical for the operative field but a radiation canopy has proved to be quite effective and is very practical.[1,12]

Fig. 5. (No. 77533.) *X*-ray of the chest after the right pleural cavity had been obliterated as described under Figures 1 through 4.

Daily cultures of the air will demonstrate periods of rising or falling air contamination and the type of bacteria present, thus aiding in anticipating the periods of greatest danger to large clean wounds.[3,7]

While these measures are all important, with the exception of radiation, they are not sufficient to give a reasonable guarantee against infection from air-borne contaminants. Killing the bacteria in the air seems to be the only feasible method of curbing this danger, and this can be done with ultraviolet radiation (2537 Å) which has a high bactericidal and low erythermal or

burning effect. The effectiveness of this addition to aseptic technic is demonstrated by the following facts:

1. Culture plates of blood agar inoculated with pathogenic bacteria can be sterilized at the operative site with an exposure of two to five minutes[9] to an intensity of radiation which will not produce evident damage to the tissues[8] or the suitably protected personnel during the course of even prolonged operations.[11,12]

2. Sedimenting viable bacteria at the operative site can be reduced by 95 to 99+ per cent by this same intensity of radiation.[1]

3. The few bacteria which survive such an exposure seem to be attenuated.

4. Unexplained infections in primary thoracoplasty incisions in the Duke Hospital were reduced by over 99 per cent.[12]

5. Infections in reopened thoracoplasty wounds in the Duke Hospital were reduced by 75 per cent.[12]

6. Deaths from unexplained infections in large wounds (thoracoplasty, craniotomy, arthoplasty, etc.) in the Duke Hospital were reduced from 1.07 to 0.00 per cent.[12]

7. Unexplained severe infections in thoracoplasty incisions (all stages) at the State Sanatorium at Sanatorium, North Carolina, were reduced from 22.7 to 1.09 per cent* simultaneously with the installation of bactericidal radiation.[12] The only infection they had in the ninety-two thoracoplasty stages since the installation of radiation was in a wound reopened for the second stage. Such a wound, of course, has sources of infection other than the air which are not present in the primary incision.

The number of bacteria in a wound at the time of closure can be reduced by washing. This is strongly advocated by some but in our hands before installation of radiation it was at the best only partly satisfactory.

In recent years, the sulfonamides are being widely used in wounds that are potentially contaminated. Naturally, they have been tried in thoracoplasty wounds but we have not used them since infections in such wounds in our operating rooms had already been practically eliminated by ultraviolet radiation. According to personal communications favorable results have been obtained by those who have used them. Undoubtedly, published results will soon appear in the literature.

Every thoracoplasty wound because of its character and size is potentially infected, and the infection rate is higher than the average surgical wound made under similar conditions. The decision as to whether or not to use the sulfonamides must be left to the judgment of the individual operator, since this decision must in the last analysis be based on his previous experience and his resultant judgment as to the possibility of infection developing in any individual wound. Where there has been a source of gross contamination, whether from a torn glove with loss of perspiration in the wound, from the air during a period of high contamination with highly pathogenic bacteria or from any other source, such a drug should be used. In view of the additional sources of contamination of reopened wounds, it is possible that one of the sulfonamides should be used even where there has been no break in technic and where the air as a source of contamination has been eliminated. This entire problem will be much clarified with additional years of experience.

Treatment of the infected thoracoplasty wound does not differ from similar infections in other wounds except insofar as the wounds differ. Naturally, infection in such a wound may be extensive and serious. They are treated by adequate drainage, chemotherapy, multiple transfusions if indicated, specific serum if such is available, and general supportive measures.

TREATMENT OF THE CAVITY FOLLOWING LOBECTOMY AND PNEUMONECTOMY

These wounds are potentially infected, but if infection can be avoided by careful

* Monroe, Clement R. Personal communication, 1941.

handling of the bronchus and meticulous technic, the operation is simplified, the convalescence is shortened, the safety of the operation is increased and postoperative deformity is reduced. Every part of the wound should be carefully protected when the bronchus is cut across, the stump should be carbolized, closed and buried beneath a flap of tissue with an adequate blood supply where this is possible. The avoidance of a bronchial fistula is imperative when an unobliterated dead space is to be left, and simplifies the convalescence in those cases in which infection occurs and obliteration of the dead space is necessary for healing.

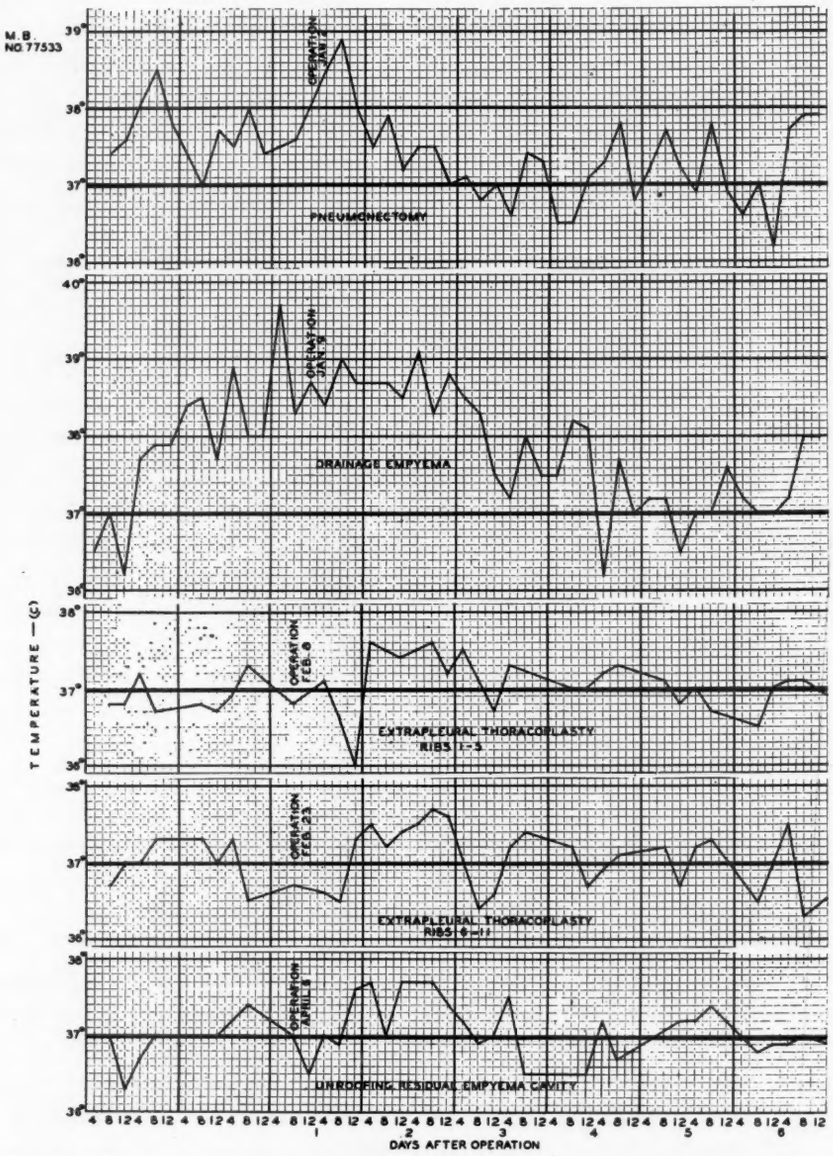

Fig. 6. (No. 77533.) Temperature curves for two days preceding and five days following each of the operations described above, Figures 1 through 4. Particular attention is called to the fact that there was a rapid drop of the temperature elevation following removal of the infected lung and again following drainage of the empyema cavity. Throughout the remainder of the patient's period of hospitalization with three operations (first- and second-stage extrapleural thoracoplasty, and unroofing of residual empyema cavity) there was no elevation of temperature on these charts or in the intervals not shown here above 37.7°C. (99.9°F.) and after each of these operations the temperature was down to normal within forty-eight hours.

If there is a reasonable chance that infection can be avoided, the wound may be closed without drainage, and one of the sulfonamides may or may not be left in the wound according to the judgment of the operator. The condition of the patient, however, must be followed closely for evidence of infection or increasing intrathoracic pressure. If either is suspected, a needle should be inserted to determine the exact condition present and if there is increased tension this can be relieved. When infection develops in the cavity, closed drainage and irrigation should be instituted. For the cavity following a lobectomy, the tube should be inserted in the dependent part of the cavity in the posterior axillary line, since this will give dependent drainage and at the same time permit the patient to lie flat on the back in comfort and without obstructing the drainage tube. If a bronchial fistula develops, suction can be maintained until the cavity is obliterated to a fistulous tract which will close when the tube is removed.

In the case of intrapleural infection following a pneumonectomy performed through an anterior incision, it is our policy to insert the tube for closed drainage through the anterior chest wall in the lower part of the cavity so as not to interfere with an extrapleural paravertebral thoracoplasty to be performed subsequently in an aseptic field. The tube can be inserted to such a depth as to irrigate from the dependent part of the cavity; and if there is a bronchial fistula, the pus can be aspirated from the most dependent part of the cavity so as to keep it empty. Under the latter condition with a large fistula negative pressure within the cavity may not be obtainable, and if the tube should be inserted into the pleural cavity below the fluid level, pus may find its way by gravity along the sinus tract about the tube. However, with the tube inserted through the anterior chest wall above the fluid level there can be no leakage of fluid along this sinus tract. Subsequent treatment may then be divided into the following stages:

1. Continuous irrigation either with an antiseptic solution or a nonirritating solution is carried on to keep the infection under control and to limit absorption. (Figs. 1, 2 and 6.) In the presence of a bronchial fistula we must be content with continuous suction and aspiration of the pus from the dependent part of the cavity along with intermittent washing out of the cavity with nonirritating fluid in quantities insufficient to flood the open bronchus. Under either condition free drainage can be obtained with a drop in the temperature reaction. (Fig. 6.) This should be the principal treatment until the acute infection has subsided. If there is infection in the chest wall incision, it may drain into the pleural cavity or to the surface, or both. Occasionally, such an incision may break down to give a sucking wound; and if this occurs, drainage can be allowed and, in the absence of a bronchial fistula, suction on the cavity maintained by covering the

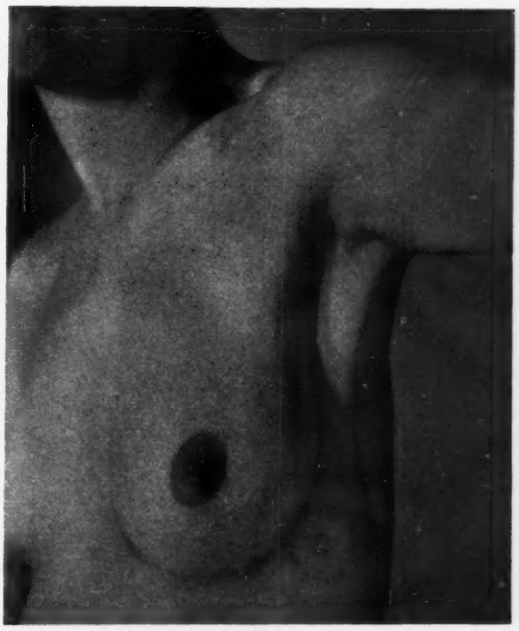

Fig. 7. (No. 77533.) Photograph showing beneath the breast the skin incision for the pneumonectomy. The breast was reflected upward and the chest entered through the third interspace. In the midaxillary line can be seen the scar from the incision made for unroofing the residual cavity after the pleural space had been collapsed to a large extent by suction and the extrapleural thoracoplasty.

wound with a sheet of thin rubber. Dental dam serves this purpose well. The wound should be supported to limit the amount of separation. In our experience infection with breaking down of the chest wall incision is rare as compared to infection within the pleural cavity.

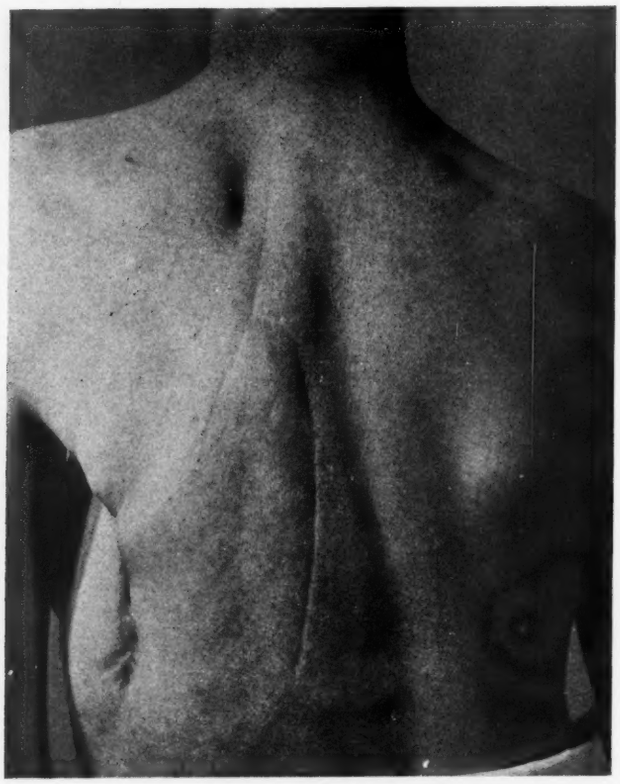

Fig. 8. (No. 77533.) Photograph showing the scars from the extrapleural thoracoplasty performed in two stages without infection (see Fig. 6) by the usual aseptic technic in a field of bactericidal radiation, while the infected pleural cavity was being drained by closed tidal irrigation. In the meantime, the cavity was being obliterated by pulling the surfaces into apposition by suction. See Figure 6 for the temperature reaction following these operations.

2. After the infection has been brought under control suction is applied to obliterate the cavity. (Fig. 2.) Following a lobectomy, this is accomplished by further expansion of the remaining lung, elevation of the diaphragm, shift of the mediastinum and partial collapse of the chest wall with a narrowing of the intercostal spaces and a greater obliquity of the ribs. Following a pneumonectomy, there is no remaining lung on the operative side to expand but the other factors all come into play with a shift of the mediastinum and increased expansion of the opposite lung. (Fig. 2.) During a pneumonectomy, every effort should be made to avoid a bronchial fistula since its presence makes the application of continuous and constant suction impossible, thus losing the great advantage of suction in obliterating the cavity.

3. While free drainage is provided by continuous irrigation and suction through a tube inserted anteriorly and leakage of pus about the tube is insured against by negative pressure within the thorax, or the location of the opening in the chest wall above the fluid level, the remaining cavity resulting from a pneumonectomy is obliterated by sucking the clean granulating surfaces into apposition as the chest wall is mobilized by an extrapleural, paravertebral thoracoplasty performed in suitable stages. (Figs. 3–8.)

4. The small residual cavity may be treated by open drainage or by complete

unroofing, with the soft parts of the chest wall falling into the cavity. (Figs. 4–7.)

CONCLUSIONS

1. The problem of wound sepsis in thoracic surgery can be largely solved for the clean primary incisions by the use of the most carefully controlled *atraumatic* technic along with the generally accepted *aseptic* technic and with the *addition* of *suitable measures* to *control wound contamination* by *air-borne pathogenic bacteria.*

2. If wound infection develops in the thoracoplasty incision, it is treated as any other wound infection except insofar as the treatment is influenced by the chronic illness of the patient, the size of the wound and the resultant seriousness of the condition.

3. Contaminated and potentially infected wounds should be treated as the clean wound, but with every additional precaution to avoid spreading the contamination and with one of the sulfonamides in the wound and in the blood to inhibit bacterial growth.

4. In the intrapleural operations every effort should be made to avoid infection and if this develops it should be drained through a stab wound and every precaution taken to avoid a breaking down of the larger incision.

5. In lobectomies and particularly in pneumonectomies every precaution should be taken to avoid a bronchial fistula. The end of the divided and closed bronchus should be covered if possible with a flap of tissue with an adequate blood supply which will grow into position before the ligatures and sutures cut through.

6. The pleural cavity infected following a lobectomy can usually be obliterated by suction alone or with the addition of open drainage of the sinus tract or the small residual cavity.

7. A fistula into an open bronchus will close if the cavity can be obliterated to a long fistulous tract.

8. The infected hemothorax following a pneumonectomy can be treated and closed by continuous irrigation through a tube inserted anteriorly out of the field of an extrapleural paravertebral thoracoplasty to control the infection, by suction to obliterate the dead space as the chest wall is mobilized by an extrapleural paravertebral thoracoplasty performed in suitable stages in an aseptic field, and by open drainage or unroofing of the small residual cavity.

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DIAPHRAGMATIC HERNIA

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ALTHOUGH many articles have been written on the subject of diaphragmatic hernia, it continues to be of interest. This interest extends alike to the general practitioner and to many of the specialists. Symptoms resulting from the condition may take the patient to the internist, gastroenterologist, cardiologist, phthisiologist, general or thoracic surgeon, pediatrician or roentgenologist. All must, therefore, be familiar with its symptomatology, its various types and the methods of treatment for each. Many of the small hiatus herniae are in all probability only incidental findings of no clinical importance and the physician must judge which are to be subjected to surgical treatment, which may be treated by medical measures and which may be disregarded. The experience of recent years has shown that the patient with a congenital hernia should be treated surgically, and authorities[1,2] are now agreed that the repair should be carried out as soon as possible.

The condition of diaphragmatic hernia is not rare and the roentgenologist in a general hospital of 300 or more beds may easily see fifteen cases each year. A recent report from the New York Hospital[3] shows an incidence of 2 per cent of diaphragmatic herniae in 4,400 consecutive examinations of the gastrointestinal tract. The incidence of the various types of herniae depends somewhat on the age and type of patient making up the hospital population. With an active pediatric service there will be an appreciable number of the interestingly variable group of congenital herniae, whereas in the hospital which treats large numbers of patients in the cancer age there is a relatively high incidence of hiatus herniae. With an active service of traumatic surgery in an industrial community the number of traumatic herniae may be large. Age, however, is not a trustworthy guide in the differential diagnosis and an occasional patient may live to a ripe, old age with a congenital hernia and without significant symptoms referable to the respiratory or intestinal tracts.

CLASSIFICATION

Diaphragmatic herniae and the associated abnormalities of the intestinal tract and diaphragm may be conveniently classified according to the accompanying schema. It should be noted that the complete and partial thoracic stomachs are not properly referred to as herniae since the term, "hernia," requires that a once normally placed organ be shifted so that it protrudes through the wall of the cavity normally containing it. The thoracic stomach was formed above the diaphragm and has remained permanently in that position because of the abnormally short esophagus.

The classification includes no indication of whether the hernia is true or false. The presence or absence of a serous sac is of some interest but knowledge concerning this feature can almost never be obtained with certainty prior to operation.

1. Thoracic Stomach
   - Entire stomach above the diaphragm.
   - Esophagus very short, ending at about the level of the fourth thoracic vertebra.
2. Short Esophagus with Partial Thoracic Stomach
   - Part of stomach permanently above the diaphragm.
   - Esophagus ends at level of seventh or eighth thoracic vertebra.
3. Hiatus Hernia
   - All or part of stomach herniated through hiatus esophageus.
   - Esophagus of normal length.

4. Congenital Hernia
   Most common through the foramen of Bochdalek, but may occur through defects elsewhere in the diaphragm.
   Large and small bowel usually included; other organs frequently.
5. Traumatic Hernia
   May occur through any part of diaphragm.
   Organs involved similar to those of congenital type.
6. Eventration of Diaphragm
   Diaphragm high but intact.
7. Congenital Absence of Diaphragm

ANATOMY

The development and structure of the diaphragm has been described in considerable detail in a recent paper by Harrington[1] but may be reviewed briefly here. The incompletely formed diaphragm may be identified in the embryo of about 7 mm. length and while the embryo grows to about 17 mm. in length this incomplete diaphragm moves caudally at a rate somewhat greater than the rate of elongation of the upper part of the enteric tube. As a result of this difference in rate of growth of the two structures the stomach is for a time partly above the diaphragm. The elongation of the esophagus begins when the embryo is about 10 to 12 mm. in length and the stomach is normally below the diaphragm when the latter structure is completely formed at the 20 mm. stage. Closure of the right leaf of the diaphragm is complete prior to closure of the left, thus accounting in part for the higher incidence of congenital herniae on the left side.

The muscle fibers make their first appearance in the diaphragm only after its structure is otherwise complete. These muscle fibers do not make up a continuous band around the periphery of the organ, but are plainly divided into three groups according to their sites of origin. The pars sternalis is a narrow band arising from the posterior aspect of the xiphoid process, and the line of separation between this band and the much larger pars costalis is usually clearly seen even in the adult. This line of separation marks the embryologic foramen of Morgagni and through it passes the superior epigastric artery (a branch of the internal mammary artery). The pars costalis arises from the costal cartilages of the six lower ribs. The pars lumbalis arises posteriorly from the crura and from the medial and lateral lumbocostal arches. The crura arise from the anterior and lateral aspects of the first three lumbar vertebrae. The medial lumbocostal arch is a fibrous band which extends from the body of the second lumbar vertebra to the transverse process of the first and bridges the psoas muscle. The lateral lumbocostal arch extends from the transverse process of the first lumbar vertebra to the twelfth rib and bridges the quadratus lumborum muscle. The line of separation between the pars lumbalis and the pars costalis is the site of the embryologic pleuroperitoneal hiatus or foramen of Bochdalek.

These three segments of musculature are frequently not solidly fused even in the adult, and the narrow zones which may separate the segments are made up only of a thin layer of connective tissue, covered above and below by pleura and peritoneum. The zones of connective tissue are much less resistant to pressure than are the adjoining segments of muscle and hence are frequently the sites of herniae.

Of the several openings in the diaphragm through which structures normally pass, that of the aorta is most posterior and is at the level of the first lumbar vertebra. This opening is not properly within the diaphragm but its anterior and lateral boundaries are formed by the crura as they extend forward from their points of origin on the spine. The thoracic duct and the azygos vein pass through the opening closely adjacent to the aorta. The opening is well bounded by dense fibrous tissue and is not a site of hernia formation. The esophageal opening is somewhat more anterior and is at the level of the tenth thoracic vertebra. Besides the esophagus it transmits also the esophageal vessels and the vagus nerves. It is entirely within the

muscular portion of the diaphragm, being surrounded by the decussating bundles of the crura before they spread out into the central tendon. There is considerable variation in the size of this opening and the space not occupied by the structures passing through it is filled by loose areolar tissue. The muscle fibers of the crura do not attach to the esophagus and some mobility of the structures is thus permitted during respiration and deglutition. It seems quite probable that the hiatus herniae so frequently seen in older people are the result of relaxation of this connective tissue. The opening for the vena cava is situated still farther anteriorly and at a still higher level, being opposite the eighth thoracic disc. It is within the central tendon of the diaphragm just to the right of the midline and is well protected by the liver.

THORACIC STOMACH

In cases of true thoracic stomach all or nearly all of the stomach is above the diaphragm, being held there as a result of extreme shortness of the esophagus. The group of such cases is very small but the term, "thoracic stomach," should be reserved for this small group and should not be used for the large number of hiatus herniae in which a relatively large portion of the stomach may be within the chest but in which the esophagus is of normal length. The true thoracic stomach is the result of an embryologic fault in which the esophagus is very short and terminates usually at or near the level of the aortic arch. The term, "hernia," may not properly be applied to this small group of cases since the stomach was not at any time in its normal position in the abdominal cavity. The classic case to which the name was originally given is that of Bailey.[4] It is of interest that his patient lived to be seventy-seven years of age and apparently had no significant symptoms referable to his gastrointestinal tract.

In the cases so far reported the stomach has been invested in a serous sac which communicated with the peritoneal cavity, but in no case has any other abdominal organ been involved.

SHORT ESOPHAGUS WITH PARTIAL THORACIC STOMACH

There is a second and somewhat larger group of cases in which there is also shortening of the esophagus, and in which a part of the stomach must, therefore, remain within the thorax at all times. This second group, however, differs from the preceding group in that the degree of shortening of the esophagus is not so marked, and as a result only about one-third to one-half of the stomach lies above the diaphragm. The group is of considerable interest and appears to be clearly differentiated from the small group of thoracic stomachs. The very short esophagus associated with the true thoracic stomach terminates at about the level of the fourth thoracic vertebra, whereas in this second group the termination is almost always opposite the seventh or eighth thoracic vertebra. Kelly[5,6] has been especially interested in the problem of the short esophagus and has called attention to the constancy of the level at which it enters the stomach. Each of the four cases seen by the author has shown the esophagogastric junction to be opposite the seventh or eighth thoracic vertebra, and in each there was definite constriction at the junction.

In the earlier studies of this group of cases the shortening of the esophagus was thought to be the result of anomalous development, but more recently it has been suggested[6,7] that the shortening might be either congenital or acquired. Many of the cases show definite constriction at the junction of the esophagus and stomach and in some this constriction has extended for a distance of 1 to 2 cm. In a few, actual ulceration of the mucosa has been noted at the junction.[8,9,10] The constriction either with or without actual ulceration at the time of study, has suggested that the shortening of the esophagus might be the result of an inflammatory process. In proof of this theory is the case reported by Kelly[6] in

which a definite esophagitis was found at autopsy.

It is of considerable interest that almost all of the early work in this group of cases was among patients who were thought to have strictures of the esophagus. This was true of the last two patients seen by the author. Both were elderly women who had been followed as cases of stricture for some years and had had repeated dilatation of the lower esophagus with relief. That a part of the stomach lay immediately beyond the stricture and above the diaphragm had not been known, due to the fact that barium had never been given with the patient in the supine or Trendelenburg position.

Narrowing of the esophagus, when first seen by the roentgenologist, is frequently of such a degree that only a thin trickle of barium emulsion will pass through it. This may be true even when the patient stands erect and when the hydrostatic pressure of a considerable column of fluid is exerted against the lesion. If the narrowing be of this degree and if the patient be maintained in the erect position, the thin trickle of barium will not completely fill that portion of the tube immediately beyond the lesion, but will instead gravitate to the most dependent portion of the stomach. It is, therefore, necessary to place the patient in the Trendelenburg position so that the barium may collect distal to the lesion and demonstrate the full caliber of the organ which lies there. A complete or partial thoracic stomach with a short esophagus would thus be readily differentiated from a simple stricture of the esophagus.

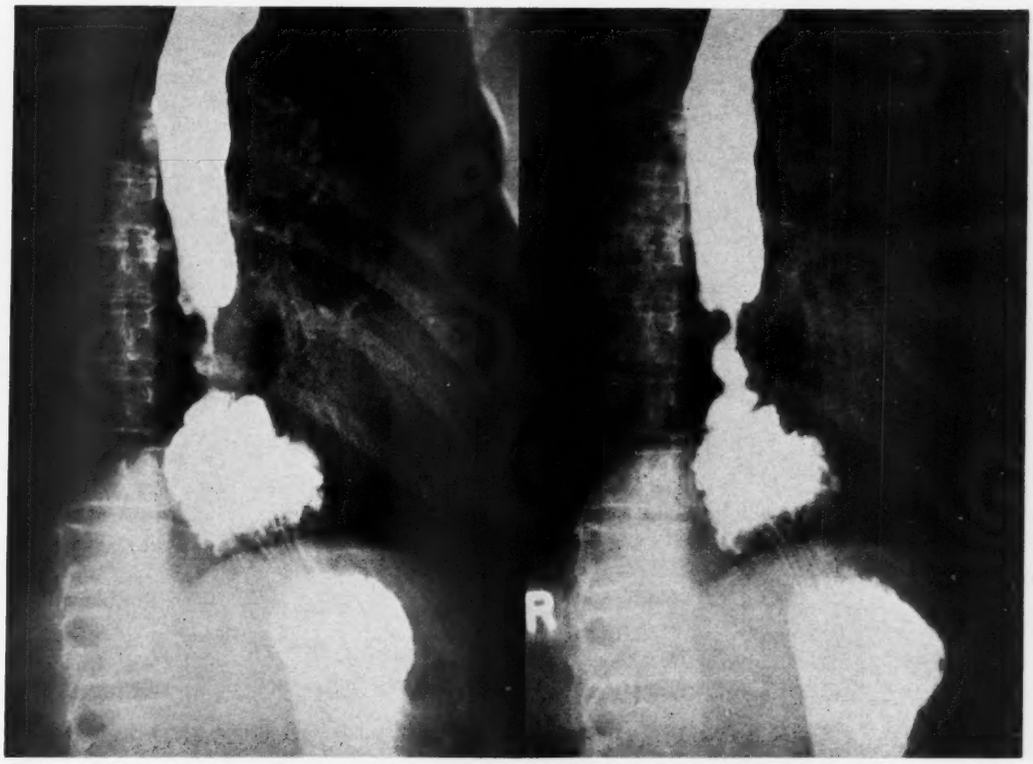

Fig. 1. Woman, seventy-five years of age. Had had difficulty in swallowing for about twenty-five years. First seen by laryngologist about fifteen years before and had been dilated repeatedly since for presumed esophageal stricture. Treatment afforded relief but it had not been appreciated that the stomach lay immediately beyond the stricture. Note that the narrowed esophagus enters the stomach at its apex.

It should be noted that in cases of short esophagus the esophagus enters the intrathoracic portion of the stomach at its apex and that the cardiac end of the stomach tends to be cone-shaped. This is in contrast to cases of hiatus hernia in which the esophagus enters the stomach from one side (usually the right posterior aspect) and

the fundus retains its usual rounded contour.

The symptomatology of the group of cases of partial thoracic stomach with short esophagus is somewhat different from that of the large group of cases of hernia through the hiatus esophageus, in which the esophagus is of normal length. The patient with the short esophageus is most apt to complain of difficulty in swallowing solid foods. As is noted below, the patient with a simple hiatus hernia is more apt to complain of a feeling of fullness after eating, of sour eructations, or of pain in the left chest simulating angina pectoris. The patient with hiatus hernia almost never complains of difficulty in swallowing. The difficulty in swallowing associated with the case of short esophagus is due to the constriction which is so often present at the esophagogastric junction.

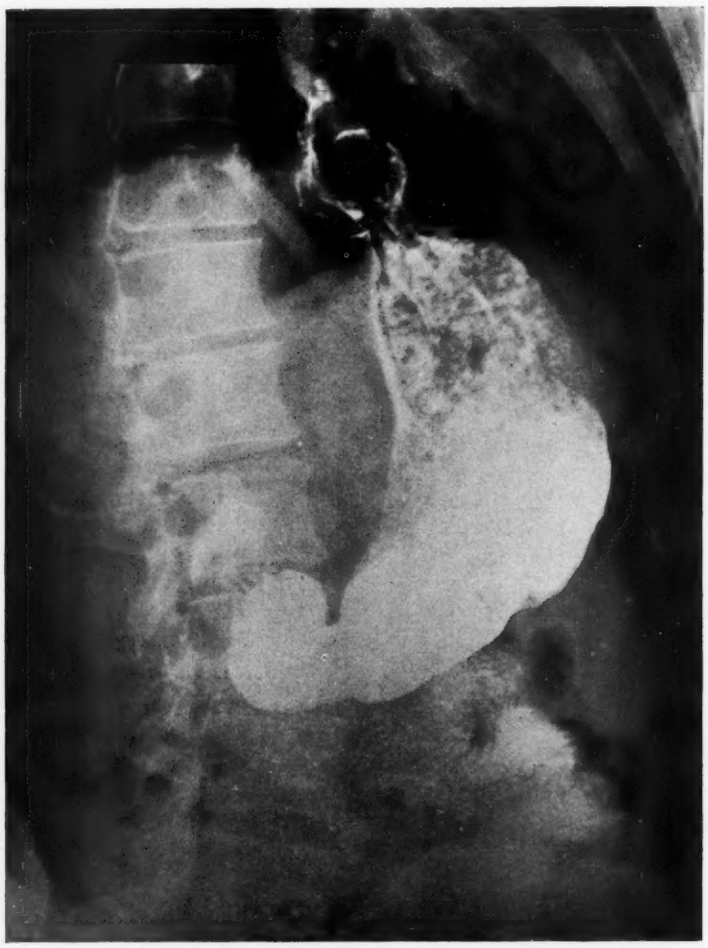

FIG. 2. Conventional Bucky film of a hiatus hernia of the "acquired" or "sliding" type. Note that the esophagus enters the stomach from the side in the same manner that it does normally when the stomach lies below the diaphragm.

Treatment of these patients, therefore, consists in dilatation of the constricted area, and such treatment usually brings about a satisfactory result. The dilatation may be combined with a modified ulcer régime directed against the inflammatory process and the tendency to ulceration. Harrington[1] has operated upon eleven patients in whom the roentgenologic diagnosis was partial thoracic stomach and short esophagus, but at operation found that the esophagus was actually short in only four. He states that in all but one patient he was able to bring the stomach below the diaphragm after separation of

the tissues around the hiatus and interruption of the left phrenic nerve. Truesdale[11] has reported one such case in which he was unable to maintain the position of the stomach below the diaphragm. This case was also referred to in a previous paper by the author,[12] whose opinion is that no attempt should be made to bring the stomach below the diaphragm in these patients. The belief that surgical repair should not be attempted in these cases makes the differentiation of this group from the hiatus group of paramount importance, since the hiatus hernia may be repaired and all symptoms referable to it relieved.

HIATUS HERNIA

Herniae through the hiatus esophageus constitute the largest group of diaphragmatic herniae. They are most frequent in persons beyond forty years of age and are more common in women than in men. The group may be subdivided depending on whether the esophagus enters the stomach above or below the diaphragm. In either case the esophagus is of normal length. In the "para-esophageal" type the lower end of the esophagus remains fixed within the hiatus and the cardiac end of the stomach herniates upward beside it. In the so-called "acquired" type, which is seen most commonly in the advanced age group, the lower end of the esophagus is not fixed but withdraws into the posterior mediastinum together with a portion of the stomach. Either of these two types may slide freely through the hiatus with change in position of the patient, or may be incarcerated. In both types the fundus of the stomach retains its normally rounded contour, and in both, the cardiac orifice is on the side rather than at the apex, as was the case with the shortened esophagus.

The hiatus hernia may vary in size from those which involve only a very small portion of the fundus of the stomach to those which involve the entire stomach. In the latter case the transverse colon may also be included as a result of traction by the gastrocolic omentum. The author has personally seen one patient (Case 4[12]) in which the fundus of the stomach lay beneath the left diaphragm and the antrum above it.

Fig. 3. Spot film of hiatus hernia.

Dr. George W. Holmes has demonstrated films of an identical case seen at the Massachusetts General Hospital. In both patients there was considerable retention of barium in both the upper and lower pouches as a result of torsion and constriction at the hiatus.

As above noted, a portion of the stomach may slide freely back and forth through the hiatus or it may be incarcerated and immobile. Incarceration is usually seen only in patients whose history extends over a period of years, and it will be noted that when incarceration is present the symptoms are almost constant. The symptoms are inconstant and change in degree with change in position when the hernia is freely reducible. If incarceration has occurred and if the hernia is therefore present in all positions and at all times, it may be noted as a gas bubble above the diaphragm and behind the heart. Its presence may thus be detected on films or fluoroscopy of the chest prior to the administration of barium.

The great majority of hiatus herniae lie chiefly to the left of the midline; but when

a large part of the stomach is involved it has a tendency to rotate on an axis through its cardiac orifice so that its greater curvature is uppermost, and it may then lie chiefly to the right of the midline.

The incidence of hiatus hernia is highest in individuals beyond the age of forty years and it has been assumed that they occur as a result of relaxation of the tissues within the hiatus esophageus. It should be noted again here that there is no attachment of the muscle fibers of the crura of the diaphragm to the lower esophagus. It is the belief of the author and of many others that the incidence of herniation through the hitaus is increased in those patients who are overweight or have relatively large abdominal tumors. Rigler and Eneboe[13] studied a series of pregnant women in whom 18 per cent of the multiparas showed herniae during the third trimester of pregnancy. Of the ten patients re-examined after delivery only three still showed their herniae. The author has seen one large hiatus hernia in a woman having a fibroid which extended to the umbilicus and has also seen two herniae during pregnancy. Of these two, one was completely gone following delivery and one was reduced to about 2 cm. in diameter.

Hiatus herniae may also be found in infants and young children. Miller, Parmelee and Sanford[14] have reported one in an infant of nineteen days, and others have also been reported in recent years.[15,16,17]

The symptoms of hiatus hernia vary in type as well as in severity and the term, "masquerader of the abdomen," which was coined by Harrington, has been adopted by many. In many patients the history is suggestive of gallbladder disease, and this history may even include sharp attacks of upper abdominal pain but no jaundice. The pain frequently goes through to the back but its point of origin should make the physician suspicious of hernia since it has a substernal component which is larger than its epigastric component. The history is frequently very suggestive of coronary disease since the distress may begin in the substernal region and radiate into the left chest and down the left arm. The final differentiation between coronary insufficiency and hiatus hernia may be quite difficult since both conditions may exist in the same patient, as reported by Moschowitz.[18]

An interesting clinical and experimental study of the rôle of the gastrointestinal tract in the production of cardiac symptoms has recently been carried out by Morrison and Swalm.[19] These authors observed that in a number of patients with angina pectoris indifferent results were obtained with the usual methods of management, but that when attention was also directed toward the gastrointestinal tract there was definite improvement. They were able to produce typical anginal pain and characteristic electrocardiographic changes by inflation of balloons in the lower esophagus and stomach and concluded that there was a reflex nerve pathway between the upper gastrointestinal tract and the heart. They, therefore, recommend frequent feedings of a bland diet together with effective doses of atropine or belladonna in patients with angina associated with gastrointestinal symptoms. Porter[20] has also reported a case in which the distribution of pain was that of angina pectoris and in which there were electrocardiographic changes associated with diaphragmatic flutter. The pain was absent and the electrocardiographic tracing normal during periods of normal function of the diaphragm.

Hiatus hernia may occasionally be associated with a secondary anemia of significant degree as earlier noted by Bock, Dulin and Brooke[21] and more recently by Harrington[1] in 11 per cent of 198 cases in which operation was performed. The anemia is usually the result of erosion of the mucous membrane associated with incarceration but may be due to actual ulceration or tumor.

Opinions vary as to the indications for surgical repair of the hiatus hernia, and it is probable that many are of little or no importance. Harrington[1] has established the general rule that they should be

repaired if more than one third of the stomach is involved and if the general condition of the patient warrants such a procedure. Repair should certainly be done if the amount of stomach involved is sufficient to permit its twisting upon itself or if the colon is also involved. In elderly patients whose general condition is poor, but in whom there is evidence of spasm of the diaphragm about the stomach, paralysis of the left phrenic nerve may give prompt relief and lasting benefit.

CONGENITAL HERNIA

The congenital herniae constitute the second largest group of diaphragmatic herniae. The group is of great interest because in it we find the greatest departure from the normal and also the greatest variation in the organs involved. They are often dramatic in their clinical course and may be the cause of death by asphyxia or as the result of intestinal obstruction.

Respiratory symptoms usually predominate over the intestinal symptoms in infants under one year of age, due to partial or complete collapse of one lung and displacement of the mediastinum. The diagnosis may be suspected within a few minutes after birth when the attention of the obstetrician or pediatrician is arrested by the abnormally weak cry of the infant. Cyanosis may also be present. Physical examination may show abnormal position of the heart with absent breath sounds on one side. These findings, either singly or combined, should be sufficient indication for roentgen examination of the chest and the suspected diagnosis may thus be immediately confirmed without the administration of barium. In children over one year of age the outstanding symptoms are usually those of recurring, partial intestinal obstruction, although dyspnea and cyanosis may continue to be present in variable degree.

It should be noted that an occasional patient with a congenital diaphragmatic hernia may live to old age without significant symptoms referable to the hernia, but it must also be noted that such a patient may live for years without symptoms and later die as a result of the lesion. Such a case is that reported by Stoll.[22]

The most common site of congenital hernia is through a persistent left pleuroperitoneal canal, or foramen of Bochdalek, the left-sided herniae being approximately ten times more frequent than those on the right. Herniae through this opening are almost always of the false variety and thus suggest that the misplaced organs occupied this abnormal position prior to the completion of the membrane dividing the celomic cavity. The major portion of the small intestine and at least half of the large bowel is usually involved. The terminal ileum and cecum are frequently to be found within the chest, thus showing an associated incomplete rotation of the colon.

Less commonly, herniation occurs through the parasternal foramen of Morgagni and it is of interest that these are almost always (perhaps always) true herniae. The organs involved usually include only a segment of the transverse colon and omentum, but the antrum of the stomach has also been described in the opening.

Occasionally, herniae occur through other parts of the diaphragm, and they may open into the pericardial cavity.[23] Ladd[24] has described a case in which congenital hernia was associated with absence of the pericardium and Ochsner[25] has operated upon a patient in whom there was absence of the anterior mediastinum. Ladd has remarked that it is surprising that associated anomalies of the pericardium and diaphragm are not more frequent in view of their intimate developmental connection.

The great majority of congenital herniae involve one or more hollow organs and the correct diagnosis may thus be strongly suspected on films made without the use of opaque media. Solid organs, however, may also be involved, and in herniae occurring through the left side the spleen is usually lodged above the diaphragm. This causes no difficulty in diagnosis, but when only solid organs are involved, for example the

liver, the diagnosis is very difficult and the homogeneous area of density at the base of the lung may be interpreted as a collection of fluid.

In the past there was a tendency to postpone any attempt at operative repair of congenital diaphragmatic hernia until the child had reached the age of several years. Experience, however, has shown that the child with a large diaphragmatic hernia remains poorly nourished and underdeveloped even though he may be free from obvious respiratory embarrassment or evidence of definite intestinal obstruction. Ladd and Gross[2] believe that the policy of waiting has been responsible for the loss of many lives and they have shown that infants in the first forty-eight hours of life stand major surgical procedures extremely well. These same authors are strongly in favor of roentgen examination to confirm the history and physical findings, but point out that in many cases all of the necessary information may be obtained without the use of barium. An added risk of obstruction is thus avoided. Harrington[26] is likewise in favor of early operation in all cases in which the large or small bowel is involved. I have been impressed by the poor state of development and general health of several children in whom operative repair had been delayed, and have witnessed the rapid return to robust health after successful operation.

These views are, therefore, in contrast to those of Kerr and Steinberg[27] who state that "Interest in this condition is more academic than practical as far as the physician is concerned except, of course, when thoracentesis is contemplated."

TRAUMATIC HERNIA

Diaphragmatic herniae due to trauma are always of the false type and are similar to the congenital herniae as regards the variability of site and of organs involved. Hedblom[28] collected a large series of cases and found that about 90 per cent occurred in males and that 50 per cent were due to penetrating injuries. It seems likely that the number resulting from crushing injuries may be found to increase with the increase in automobile accidents.

The opening in the diaphragm usually extends the full length of the muscle fibers and runs radially from the central tendon to the chest wall. The tendency of the opening to be of this slit-like character is seen even in those cases in which the original injury was that of a small object, such as a rifle bullet. In these cases it seems reasonable to believe that the originally small opening enlarges along the course of the muscle fibers as a result of repeated passage of the abdominal organs through the opening.

The raw edges of the opening are conducive to the formation of adhesions and it is, therefore, understandable that the incidence of intestinal obstruction is higher in traumatic herniae than in any other group of diaphragmatic herniae. Operative repair is, therefore, indicated in all cases unless the general condition of the patient does not warrant such a procedure. Delay is hazardous since the operative mortality is greatly increased in the event of intestinal obstruction.

EVENTRATION OF THE DIAPHRAGM

Eventration of the diaphragm should be included in any discussion of diaphragmatic herniae since the differential diagnosis is often a difficult one. The condition is much more common on the left side than on the right and is rarely bilateral. The high but intact diaphragm may or may not be paralyzed and its motion may be normal, diminished, absent or reversed.[29]

Satisfactory roentgen demonstration of the elevated diaphragm is at times very difficult, although it is normally possible in the right anterior oblique or right lateral positions. A number of procedures have been suggested for demonstrating the diaphragm and for differentiating eventration from hernia but none has been found to be entirely trustworthy.

As a rule the symptoms resulting from eventration are not severe and the diagno-

sis is suspected only because of the abnormal findings on physical examination. A few patients experience distress in the region of the left costal margin due to collection of gas in the stomach and colon. It will be noted that the fundus of the stomach may be well above its cardiac orifice in these cases and that gas may thus be effectively trapped there.

Since the symptoms do not as a rule warrant such a procedure only a few attempts have been made to correct the deformity. Correction by plication of the relaxed diaphragm does, however, seem possible.

CONGENITAL ABSENCE OF THE DIAPHRAGM

Congenital absence of the diaphragm is an even rarer condition but one which must also be considered. That the condition is not incompatible with life is shown by the patient reported by Harris and Greene.[30] Their patient was thirty-one years of age and had two children. The pressure relationships between the abdomen and left hemithorax in such a case are definitely abnormal but are identical with those of the patient with a large false hernia of the congenital or traumatic type. The condition may thus be embarrassing and is of course not curable but is nevertheless compatible with life.

The only merit in including the rare anomaly of congenital absence with the relatively common condition of diaphragmatic hernia is to point out that the diagnosis is not possible except after careful exploration by a competent surgeon. It must be borne in mind that in many cases of congenital and traumatic hernia the remnants of the diaphragm cannot be demonstrated by roentgen examination, and yet in such cases sufficient diaphragm may be found to effect a satisfactory repair in almost all cases. Careful exploration should, therefore, be carried out in all suspected cases, since the danger of obstruction is a very real one if the condition is actually that of hernia.

SUMMARY

Diaphragmatic hernia is an uncommon condition but is met with in almost all branches of the practice of medicine.

The complete thoracic stomach is an extremely rare anomaly and apparently gives rise to few symptoms. The partial thoracic stomach with shortening of the esophagus gives rise to symptoms which are most often the result of constriction at the junction of esophagus and stomach. It is important that both of these conditions be differentiated from the much more common hiatus hernia, in which the esophagus is of normal length and in which satisfactory surgical repair may be done.

Early operation is recommended for all cases of congenital and traumatic herniae because of the danger of intestinal obstruction.

Congenital absence of the diaphragm may not be diagnosed prior to careful exploration.

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DEFORMITIES OF THE CHEST AND SPINE RESULTING FROM THORACIC DISEASE AND OPERATION: THEIR PREVENTION

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IT is not enough merely to cure a disease. Always, every effort should be made to restore a patient to as nearly a normal condition as possible. That is to say, a cure should be accomplished with the least possible functional, cosmetic and economic sacrifice.

Too often, as a result of specialization there has developed among surgeons a tendency to become somewhat thoughtless of the fact that the human organism is an intimately integrated functional unit. Thoracic surgeons as a group are no exception. In their zeal to improve or put right the organs within the chest they not infrequently fail to appreciate the importance of the thoracic spine as an integral part of the thoracic cage. As a result not a few unnecessarily disfiguring and crippling deformities have been permitted to develop and unfortunately once deformities have become established they are difficult or impossible to correct.

The preventable deformities with which we are concerned are the rotational and lateral curvatures of the spine which may be grouped under the caption of thoracogenic scoliosis. These are of two types, pleurogenic or empyema, and thoracoplastogenic or thorocoplasty scolioses. Since an understanding of the differences in the two types of curvatures and in the mechanisms of their production is essential to intelligent preventive and corrective management, they will be discussed in some detail.

The pleurogenic type is produced mainly by forces which act from within the chest and which are abnormal, namely, contraction of pleural scar, while thoracoplasty scolioses arise from forces which normally act upon the spine, in which forces, however, a disturbance of equilibrium has taken place.

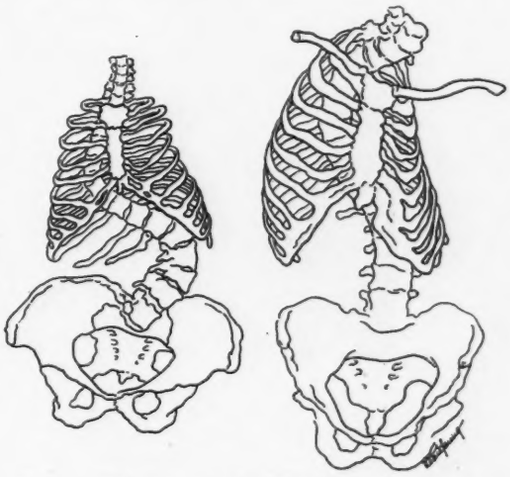
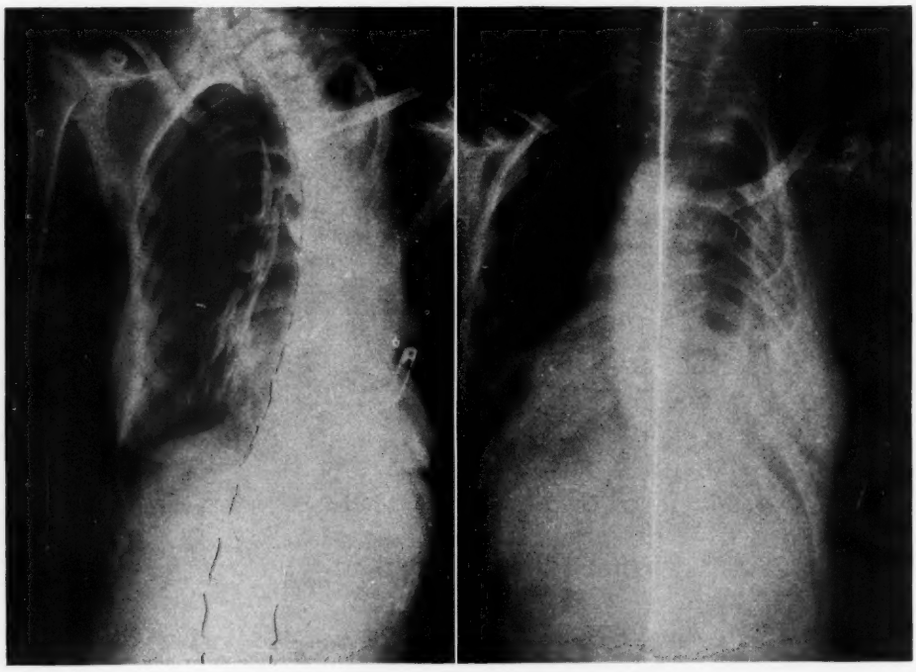


FIG. 1. Diagrammatic drawings for a comparative analysis of rotatory and nonrotatory scoliosis. To the left is illustrated the rotation deformity of the thorax of idiopathic scoliosis, which typifies the rotatory type of scoliosis. The drawing to the right represents the deformity of pleural scoliosis which uniquely is a nonrotatory type of curvature. (From Bisgard, J. D. Thoracogenic scoliosis. *Arch. Surg.*, 29: 417, 1934.)

The normal thoracic spine is maintained in a state of static equilibrium by a balance of the opposing forces acting on the two sides of the vertebrae, and the ribs are the principal agents of support by virtue of their rigidity not only as bony struts but also as instruments through which the forces exerted by the attached muscles and ligaments and the forces of intrathoracic pressures and stresses are transmitted to the vertebrae through their costovertebral articulations. Thus, normally, each pair of ribs grips with pressure the vertebrae with which it articulates; and if the pressure

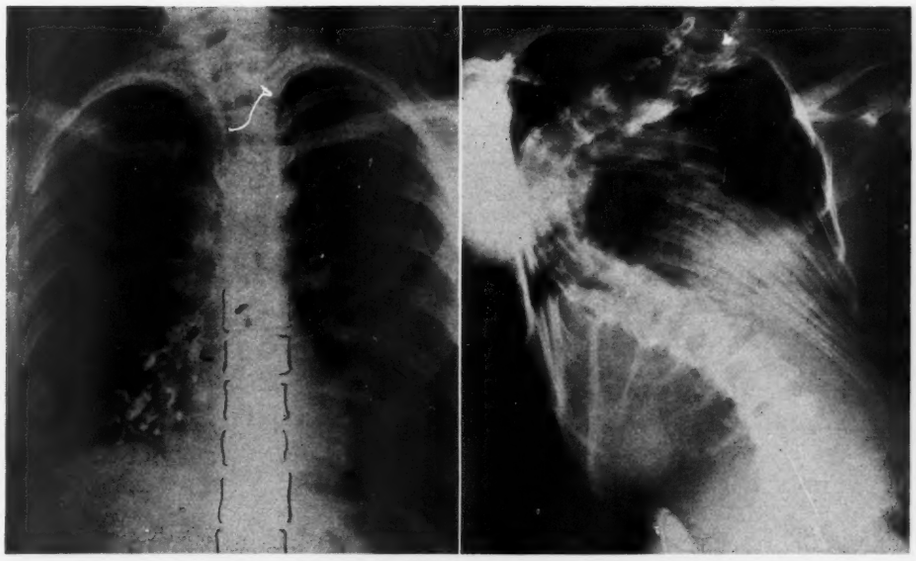
is unequal on the two sides or if it has been removed from one side by the extensive resection of ribs, such as occurs in thoracoplastic operations, the unopposed ribs push the vertebrae toward the unsupported side and a deviation results. A spine which was straight before operation will deviate with the convexity of the curve projecting into the side on which the operation was performed. This is the thoracoplasty type of scoliosis and results principally from removal of struts from one side of the vertebral bodies. It is a rotatory scoliosis as are all spinal curvatures with the exception of the pleurogenic type. That is, the vertebrae not only shift laterally but also rotate around the vertical axis as they deviate from it. As the vertebrae rotate they twist the costal framework so that the thoracic wall on the side of concavity, the unoperated side, becomes flattened posteriorly and bulged anteriorly. The deformity on the operated side is determined principally by the distortion resulting from the costal resection.

In the production of pleurogenic scoliosis two mechanisms are operative. First, as a result of pleural irritation and of pain there develops protective spasm of the muscles on the affected side and the spine is bent with the concavity of the curvature on that side. Thus there is an initial spastic scoliosis. Subsequently, the second and more important factor exerts its influence. The pleural scar resulting from chronic pleuritis from any cause, such as a simple serous tuberculous effusion or tuberculous or nontuberculous empyema, contracts gradually and through adherence to the ribs pulls them centripetally. Since the ribs, like bucket handles, can pivot only at their extremities (the vertebral articulations posteriorly and the cartilages anteriorly) and inscribe arcs as they are drawn mesialward, they also move downward. This force is transmitted to the



A B

Fig. 2. Roentgenograms in a case of (A), thoracoplasty scoliosis, and (B) pleurogenic scoliosis. Compare with Figure 1. Note that the thoracoplasty curvature results from herniation or protrusion of the spine into the unsupported side and as such is a deficiency deformity whereas the pleurogenic curvature results from forceful bending and is a contracture deformity. (From Bisgard, J. D. Thoracogenic scoliosis. *Arch. Surg.*, 29: 417, 1934.)



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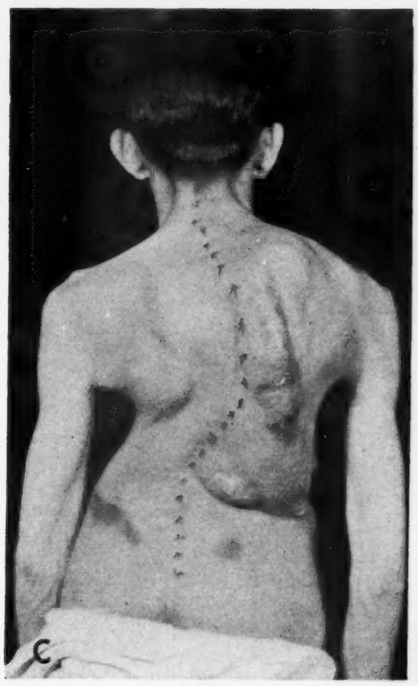


C

Fig. 3. An extensive fixed deformity which resulted from resection of ribs. A, roentgenogram showing bronchiectasis of the right lower lobe, which had been filled with iodized poppy-seed oil, 40 per cent. Note the slight sinister convex scoliosis. B, three years after total excision of the right lower lobe after removing long segments of three ribs. The spine has reversed its curvature and completely collapsed into the side on which operation occurred. Note the rotation of the vertebrae and the deformity of the thoracic wall caused by the rotation. C, photograph of the same patient illustrating the characteristic deformity of rotatory scoliosis. (From Bisgard, J. D. Thoracogenic scoliosis. *Arch. Surg.*, 29: 417, 1934.)

vertebral bodies through their costovertebral articulations, traction being exerted on the uppermost thoracic vertebrae by the upper ribs, which are pulled downward, and pressure being exerted on the lower ones, especially on those at the apex of the curve. The result is a spinal deviation with the convexity of the curve projecting into the healthy side, directed reversely to that produced by thoracoplasty.

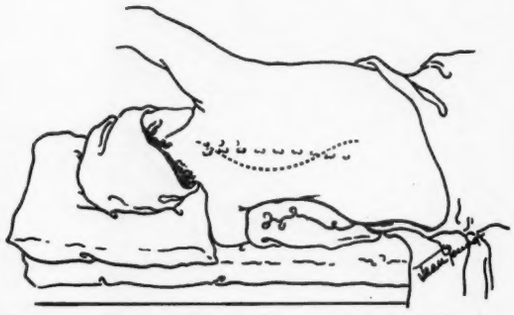


Fig. 4. Postural wedging as used following thoracoplasty. The patient lies on the diseased side, or the side on which operation was performed, over a pillow which has been rolled in the direction of its long axis and placed at right angles to the axis of the body. By placing this fulcrum properly, the spine can be wedged in the direction desired. To correct a thoracoplasty scoliosis, the wedge is placed slightly more distal, that is, directly opposite or slightly caudal to the apex of the curve. Note the change of spinal contour which can be brought about by this procedure as illustrated diagrammatically by the straight alignment of the spinous processes. The curved, broken line illustrates the position of the spine of a thoracoplasty scoliosis before wedging. (From Bisgard, J. D. Thoracogenic scoliosis. *Arch. Surg.*, 29: 417, 1934.)

The nature of the etiology of pleurogenic scoliosis makes it a distinct clinical entity. It differs from all of the other types in that there is no, or but slight, rotation of the vertebral bodies. This disobedience to the well recognized physical law of the dissipation of part of the bending force of rods into the production of rotation or twisting is due to the immobilization of the ribs in their retracted positions. The vertebrae cannot rotate against the resisting pressure exerted on them by the ribs, which are rigidly fixed by the pleural scar. This scar in certain instances undoubtedly exerts force directly on the vertebral bodies, which helps to prevent rotation and usually also fixes the mediastinum to the spine so that it deviates with the spine. Since there is little or no rotation of the vertebrae, the thoracic cage develops only the distortion which results from lateral bending. The essential differences in the rotatory and nonrotatory types of scoliosis are illustrated in Figures 1 and 2.

Although most patients with pleural disease, many with pulmonary disease and all that have had extensive costal resections, develop some curvature or change in the alignment of their thoracic vertebrae. In only a few of them, fortunately, do the curvatures progress to a stage of deformity or have any clinical importance. It should be noted also that deforming curvatures develop rarely in adults. All of the author's cases have occurred in children and adolescents. As stated in a previous publication, "Severe deformities are in most instances indictments of inadequate treatment because they are preventable. They are important, although they occur infrequently, because they are irreparable and because the extensive curvatures of the spine and distortions of the costal framework produce not only disfigurement of great concern to the patient, but also incapacity from impairment of cardiac and respiratory functions which not infrequently results in premature death." In Figure 3 is shown an extensive deformity which resulted from resection of portions of three ribs and the lower lobe of the right lung.

Disturbed anatomic relations of the heart and great vessels which impair their functional efficiency, and evidence of interference with the return flow of the pulmonary circulation with chronic passive congestion of the lungs have been observed frequently at necropsy (Eckardt 1927). These factors not only limit the patient's activities but also diminish his ability to combat any severe illness successfully. Pneumonia is a frequent cause of death.

Since it is difficult or impossible to correct established deformities, we should

concern ourselves principally with preventive therapy. Every child and every adolescent with pulmonary or pleural disease or who is undergoing operative treatment, which involves resection of portions of several ribs, should be considered a potential scoliotic and efforts to prevent deformity should be instituted immediately.

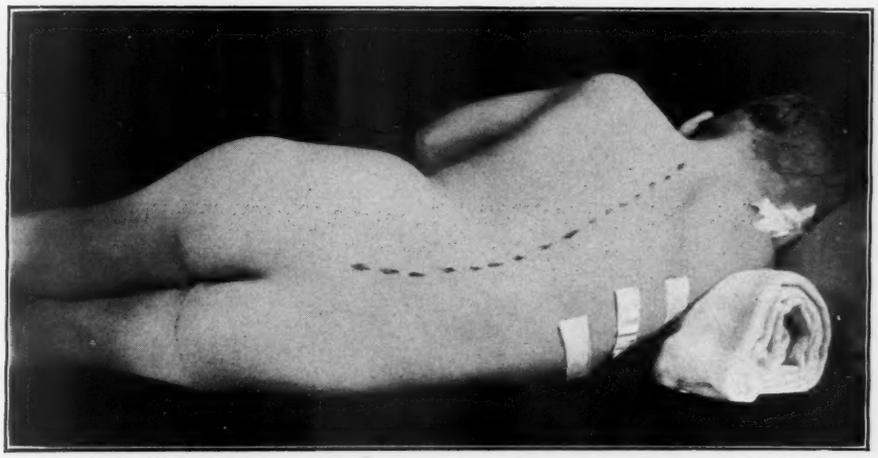


Fig. 5. Postural wedging to prevent development of a curvature in a case with empyema of the right pleural cavity. (From Bisgard, J. D. Postural wedge compression of thorax. *J. Thoracic Surg.*, 7:609, 1937.)

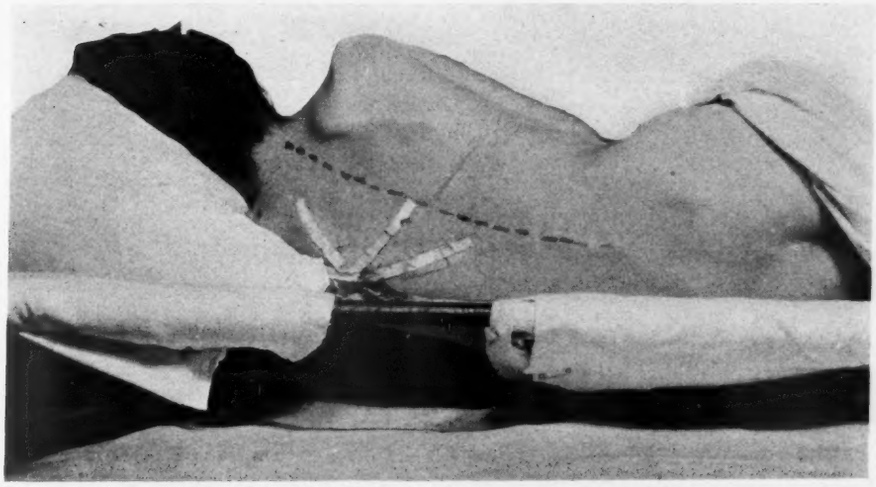


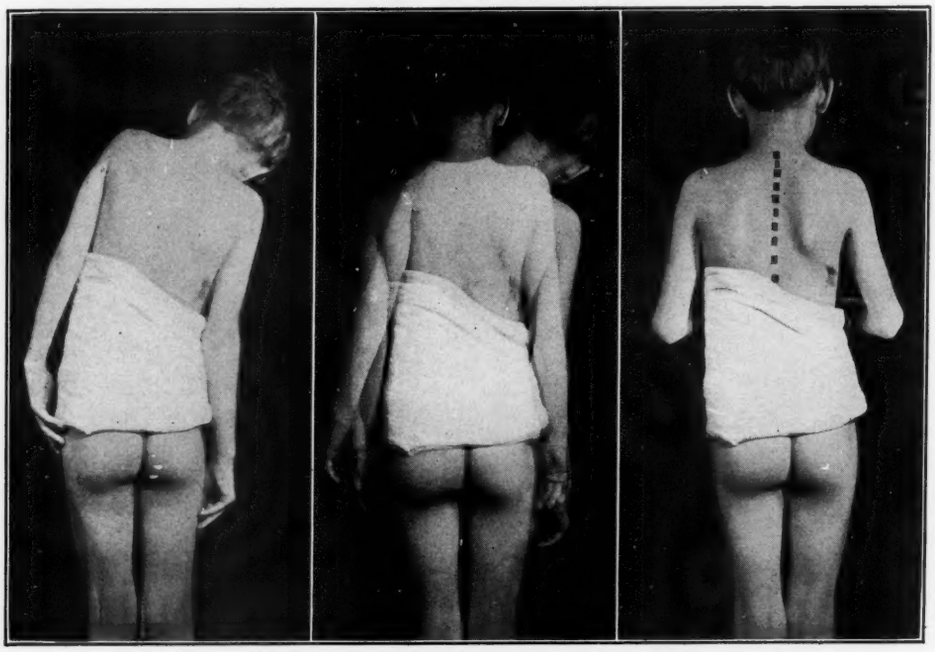
Fig. 6. A patient with a dependently drained encapsulated interlobar empyema of the left chest, lying upon a Bradford frame upon the affected side which, with the drainage tube, protrudes through the gap free from pressure. In this position drainage is dependent, the spine is bent into a position of overcorrection and the unaffected side has unrestricted motion. (From Bisgard, J. D. Postural wedge compression of thorax. *J. Thoracic Surg.*, 7: 609, 1937.)

The spinal deviations resulting from muscle spasm associated with acute nontuberculous empyema usually correct themselves when the empyema cavity is obliterated promptly. Deformities develop with the chronicity of the disease, and with a few exceptions, reflect improper treatment. The first objective in the prevention of pleural scoliosis, is therefore, the early closure of empyema cavities by early, adequate and dependent drainage. The earlier the cure of the pleural disease the less will be the accumulation of scar tissue for subsequent contracture. One may use the analogy of cutaneous burns and the contracture deformities which result from them unless the granulating surfaces are rapidly epithelized.

In pure tuberculous effusions, rapid closure of pleural cavities may be effected only by repeated thoracenteses, because drainage is contraindicated, but drainage is applicable to many tuberculous effusions which are secondarily infected by pyogenic micro-organisms.

In all cases with pleural effusions, regardless of the etiology, a constant effort should be made to hold the spine in alignment or slight overcorrection during the major portion of each twenty-four hours during the period of active treatment. In cases which have developed evidence of curvature the above treatment should be continued for several months after the pleural cavity has been obliterated, in order to prevent deformity from latent contracture of the pleural scar. With the exception of infants and unco-operative children, this can be accomplished in most patients by posture; the patient lies on his side in such a way as to deviate the spine in the direction of overcorrection sufficiently to counteract the tendency to pleural scoliosis.

The spine can be controlled and held in a corrected or overcorrected position most simply and most effectively by posture alone, or by bending the spine by postural wedging. In the latter procedure, illustrated in Figures 4 and 5, the patient lies on either side on a soft compression wedge, such as a pillow, which is rolled in the direction of the long axis and placed at right angles to the axis of the body. If this fulcrum is placed properly, the weight of the body wedges the spine in the direction desired. Preferably, postural wedging is accomplished with the patient lying upon the diseased side. The wedge is placed high in the axilla of that side (with the arm completely abducted and the head elevated) so that the entire thoracic spine is deviated in the reverse direction, the direction of overcorrection. Lying upon the diseased side is preferred because it permits dependent drainage, immobilizes that side and does not interfere with the free respiratory excursions and pulmonary ventilation of the healthy side. This position



A B C

FIG. 7 A, B and C. Plaster body cast used in ambulatory treatment. It is applied with the thoracic, or primary, curvature exaggerated so that the spine is wedged straight over the edge of the cast. (From Bisgard, J. D. Postural wedge compression of thorax. *J. Thoracic Surg.*, 7: 609, 1937.)

can be maintained (whereas it might otherwise be impossible on account of pain) by anesthetizing the wound by commodate the buttocks, is shifted toward the head end sufficiently to leave the lower half of the chest unsupported. Thus the

A

B

FIG. 8 A and B. Postural wedging. An infant three months old with empyema of the right pleural cavity. Spine held constantly in overcorrection in a plaster of Paris bed with a removable restraining lid. (From Bisgard, J. D. Postural wedge compression of thorax. *J. Thoracic Surg.*, 7: 609, 1937.)

crushing three intercostal nerves at the time of establishing drainage. It has been my practice to establish drainage by costal resection through the vertical incision advocated by Churchill. This permits easy exposure of one intercostal nerve above and one below the resected costal segment. These three nerves I routinely crush.

Several recent cases have been bedded upon Bradford frames as illustrated in Figure 6. A standard frame is suspended upon blocks in the usual manner so that it rests six or more inches above the surface of the bed. The gap between the canvas sheets, which ordinarily is placed to accommodate the buttocks, is shifted toward the head end sufficiently to leave the lower half of the chest unsupported. Thus the patient can lie comfortably and almost constantly upon the affected side, with the drainage tube protruding through the gap. With pillows under the head and shoulder, the dependent side will sag into the gap, diverting the spine into a position of overcorrection as shown in the photograph. Also with the affected side down, drainage may be made more dependent and the diseased side somewhat immobilized by the superincumbent weight, while the normal, unaffected half of the chest is given a maximum of unrestricted motion. The heart and other intrathoracic structures gravitate into the effected side, encourag-

ing obliteration of the empyema cavity and increasing the capacity for ventilation of the lung on the unaffected side.

Young children may be held in position for postural wedging in a plaster bed which is moulded to the body with the patient lying on the side in a position of overcorrection. The side walls should be high enough to prevent the child from rolling out of this position. A plaster of Paris bed is illustrated in Figure 8. Overcorrection in such a bed should be maintained for many weeks and until there is no tendency to recurrence of the curvature.

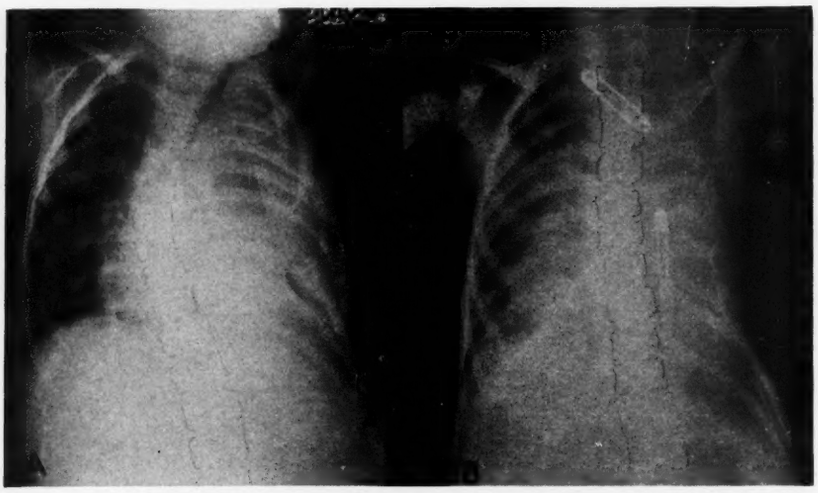


FIG. 9. A, a tuberculous empyema of two years' duration in a child of four years has been productive of a moderately severe pleural scoliosis. B, the same patient two weeks after completion of a Schede thoracoplasty. Note the correction of the pleural scoliosis by the imbalance produced by the operation. (From Bisgard, J. D. Thoracogenic scoliosis. *Arch. Surg.*, 29: 417, 1934.)

Of the methods of securing postural correction the use of the Bradford frame has proved the most satisfactory and will be used routinely by the author in the future.

If the curvature persists after the patient becomes ambulatory, correction can be accomplished and maintained by the use of a special type of plaster jacket. It is applied with the body bent to the affected side until the lumbar or secondary curvature has been corrected. The cast extends slightly below the greater trochanters on both sides and to the twelfth rib on the unaffected side. Very little padding is used, and the cast is made to fit very snugly. As the patient assumes an erect posture to establish balance, he automatically corrects the thoracic or primary curvature by wedging the spine over the edge of the cast. The principle is illustrated in Figure 7. This type of jacket should be worn for two or more months. After its removal, the patient should be seen at monthly intervals for at least a year, and with the slightest evidence of a recurrence of the curvature, the jacket or cast should be reapplied.

Although the vertebral column deviates or sags into the unsupported side of the chest after the extensive costal resections of a posterolateral thoracoplasty, the vertebrae are sufficiently plastic to permit considerable moulding and correction by force until it becomes fixed from four to eight weeks after operation by regenerated ribs, organized fibrous tissue and reinserted muscles. If correction is maintained during this reparative period, permanent curvatures will be minimized and often prevented. If, however, the periosteum has been destroyed or excised (as it is in a Schede thoracoplasty), the resected costal segments are not replaced, and the spine is permanently deprived of much of its support on the side on which operation has been performed. In many instances, how-

ever, this lack of support may compensate for the previous imbalance from the pleural scar and merely correct a preoperative pleurogenic scoliosis. Thus, as illustrated in Figure 9, the operation performed for the purpose of obliterating a chronic empyema cavity often corrects the curvature which is usually present in these cases. Not infrequently it overcorrects and replaces the pleurogenic by a thoracoplasty type of scoliosis. The postoperative prophylactic maintenance of correction by postural wedging in these cases is beneficial but less effective than in the cases in which the extrapleural type of operation has been done. It is desirable, therefore, to obliterate as much of the empyema cavity as is possible by extrapleural resection of ribs.

To prevent and correct thoracoplasty scoliosis, postural wedging should be instituted within a few days after operation and maintained for at least six weeks, or until the reparative processes have fixed the thoracic wall. The procedure is carried out with the patient lying on the operated side but with the wedge placed exactly opposite or slightly caudal to the apex of the curve, as illustrated in Figure 4.

With co-operation from the patient, postural wedge compression may be started immediately, or a day or two after operation, and within a few days may be maintained almost continuously.

Following thoracoplasty there is a tendency for the shoulder on the operated side to become elevated and if the costal resection has not been carried below the seventh rib the tip of the scapula may become caught on the ridge formed by the unresected ribs and hold the shoulder in an elevated position. This can be corrected by resecting the distal portion of the scapula.

The treatment of extensive and rigid spinal deformities belongs to the province of orthopedic surgery, so will not be discussed in this presentation. Suffice it to say that they are usually irreparable.

SUMMARY

1. Thoracogenic scolioses are of two types: pleurogenic and thoracoplastogenic. Both are preventable and are curable if corrected in their incipience. Extensive and rigid deformities of the spine and costal framework are incurable.

2. Pleurogenic deformities can be prevented by (1) the prevention of the formation of pleural scar by early cure of the empyema or pleuritis, and (2) the constant attention to maintenance of straight alignment or preferably of over correction of the spine. This can be accomplished by means of (a) postural wedging, (b) corrective body casts of plaster of Paris, and (c) resection of ribs on the side of concavity. To facilitate correction and the use of corrective measures, it is helpful to anesthetize the area of the wound by crushing the intercostal nerves which supply that area.

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SLIPPING RIB CARTILAGE

WITH REPORT OF CASES

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ABNORMAL mobility and the resulting deformity of the anterior ends of the anterior costal cartilages of the vertebrochondral ribs (the eighth, ninth and tenth on either side) causing painful symptoms and disability, have received but little attention in medical literature. Some textbooks, notably that by Key and Connell, make brief reference to this condition, but there is no general understanding as to what it is, why it occurs or how the symptoms are produced. It therefore seems wise to restate, to a large extent, my original paper, "A Study of the Slipping Rib Cartilage Syndrome," which appeared in the *New England Medical Journal*, 1941, comment anew on this symptom complex observed over a period of twenty-nine years, and make a further report of cases.

This syndrome has to do with the distal ends of the anterior rib cartilages, their interchondral articulations and the intercostal nerves. It does not involve the costochondral junction of the anterior ends of the ribs. Barring anomalies, mistakes in numbering the ribs, and exceptions, it involves only the anterior cartilages of the first three false ribs, anatomically designated vertebrochondral ribs, since their cartilages articulate anteriorly not with the sternum but with the cartilages of the ribs above. They are the eighth, ninth and tenth ribs on either side.

In the *Practitioner*, 1919, under the caption "On Various Conditions That May Simulate the Referred Pains of Visceral Disease and a Consideration of These from the Point of View of Cause and Effect," this symptom complex was first reported by Edgar F. Cyriax,[1] of the Medico-psychological Clinic, London. He specifically mentioned three cases treated expectantly.

In the *British Medical Journal*, 1922, under the original title "Slipping Rib," Davies-Colley,[2] Surgeon to Guy's Hospital, London, reported two cases of distal end anterior rib cartilage displacement which he had operated upon by resection of the loosened cartilages with "complete relief of symptoms." Davies-Colley[2] records that one of his patients had previously submitted to an exploratory laparotomy without relief of symptoms, but was immediately cured by the removal of the loosened cartilage.

Subsequently, and up to 1924, there were reported in the *British Medical Journal* under the heading "Slipping Rib," eight other cases by five authors,[3–7] three from England, one from Ireland and one from Egypt. Of these eight cases, three of the patients were operated upon with cure.

In 1931, two cases were reported in America, one by Dewey Bisgard,[8] from the University of Chicago clinics and one by J. A. Darby,[9] of Vancouver, Washington. Both of these patients were operated upon with cure.

Davies-Colley[2] noted: "It is probably not a rare condition and is a trivial enough complaint in itself but it gives rise to most irksome symptoms. . . . In its (the pain's) position at the costal margin it resembles that due to so many deeper lesions of the abdomen and thorax, that I think it is quite likely that many cases occur in which such an apparently unimportant cause as a movable rib cartilage is unsuspected and the diagnosis missed."

Slipping rib cartilage is of common occurrence, often producing symptoms that are sometimes of an incapacitating nature.

Diagnosis is made by the history and physical examination. *X*-ray is helpful, by excluding deeper maladies of the thorax and abdomen. Cure is obtained by a simple operation.

Failure to recognize this symptom syndrome has led to needless laparotimes, prolonged suffering and varying degrees of incapacity. Then, too, while the condition has been recognized, there does not appear to be any clear conception of its development. It therefore seems important to present again the primary fundamental features of this syndrome in the hope that it will lead to a more general understanding and stimulate a greater interest in the subject.

This loosening deformity involves the costochondral articulations and the anterior ends of the anterior cartilages of the vertebrochondral ribs, namely, the eighth, ninth and tenth ribs on either side. It is produced first by the pulling away of the anterior end of the cartilage, and secondly by the curling upward of the loosened end, so that on motion the deformed end rubs against the inside of the rib above, causing pain; also on certain motions and manipulation the deformed end slips over the rib border with a click that is felt by the patient, and a pain that is sometimes severe and incapacitating.

Figure 1, an anatomical diagram of the framework of the anterior chest wall from Gray's "Anatomy"[10] recalls that the anterior ends of the anterior cartilages of the eighth, ninth and tenth ribs are normally connected to those above not by cartilaginous union, but by a fibrous attachment. Less clearly shown, but easily demonstrable, is a fibrous hammock supporting the cartilage ends and enclosing the interchondral synovial membranes of the costochondral articulations. This encircling type of attachment lends to the desirable mobility of the anterior chest wall and rib border, but at the same time it has the characteristic of instability of any joint and hence the susceptability to trauma. Important here to remember in the consideration of cause and effect is a statement of Howard Lilienthal:[11] "The weakest part of the thorax is along the costo chondral line on either side." One should also have in mind, especially with reference to the development of symptoms, the various muscle origins and insertions with their divergent pull, the nerve supplies involved and the constant respiratory motions in this region.

The *intercostal muscles*, internal and external, occupy the intercostal spaces. Each arises from the lower border of the rib and costal cartilages above and are inserted into the upper border of the rib and costal cartilages below. Their fibers pass obliquely but in opposite directions, and extend from the tubercle of the rib behind to a point near the termination of the costal cartilage of the rib in front, where they end in a thin membrane. Their action is to stabilize the chest wall, particularly the intercostal spaces. Their nerve supply is from the intercostals.

Figure 2 pictures the diaphragm which arises from the back of the ensiform process, from the cartilages and adjacent portions of the six lower ribs, from the lumbocostal aponeurotic arch and from the lumbar vertebrae. From this series of origins it converges to be inserted into a central tendon. The diaphragm is the principal muscle of inspiration. During inspiration the lower ribs are fixed. Contraction of the muscle lowers the diaphragm, exerting a strong pull on the rib border. "In all expulsive acts the diaphragm is called into action to give additional power to each expulsive effort. Thus before sneezing, coughing, laughing, crying, or vomiting, and previous to the expulsion of urine or feces or of the fetus from the uterus, a deep inspiration takes place," throwing sudden and added strain on the rib borders. The nerve supply is from the phrenic and lower intercostals.

Figure 3 shows the *transversus abdominis* muscle which arises in part from the inner surfaces of the cartilages of the six lower ribs, interdigitating with the diaphragm to

be inserted into the crest of the pubes, the pectineal line and the linea alba.

Figure 4 illustrates the *obliquus internus abdominis* muscle which arises by fleshy fibers from inguinal ligaments, the crest of the ilium and the lumbodorsal fascia. It is inserted into the pubic crest, pectineal line, linea alba and the cartilages of the six lower (the false) ribs.

Figure 5 represents the *obliquus externus abdominis* muscle which "arises by eight fleshy digitations, from the external surfaces and inferior borders of the eight lower ribs and their anterior costal cartilages; the five superior serrations . . . are received between the corresponding processes of the Serratus anterior; the three lower ones . . . between corresponding processes from the Latissimus dorsi." The muscle extends downward to be inserted into the anterior half of the crest of the ilium, and, opposite a line between the ninth costal cartilage and the anterior superior iliac spine, it ends in an oponeurosis which continues, to unite with its fellow of the opposite side, covering and partly enclosing the rectus abdominis.

These abdominal muscles act in executing and stabilizing trunk positions. They function in conjunction with the diaphragm to compress the abdominal viscera in all expulsive efforts, and to compress the lower thorax in assisting expiration. "If the pelvis alone be fixed, the thorax is bent directly forward, when the muscles of both sides act, when the muscles of only one side contract, the trunk is bent toward that side and rotated toward the opposite side. If the thorax be fixed, the muscles, acting together, draw the pelvis upward, as in climbing; or, acting singly, they draw the pelvis upward, and bend the vertebral column to one side or the other." The nerve supply of the abdominal muscles comes from the lower intercostals and the lumbar plexus.

The serratus anterior, also shown in Figure 5, is a thin muscle situated between the ribs and the scapula on the lateral aspect of the chest. It arises by fleshy digitations from the outer surfaces and superior borders of the upper eight or nine ribs and rib cartilages, and from the aponeuroses covering the intervening intercostals. From this extensive attachment the fibers pass backward, are closely applied to the chest wall, reach the vertebral border of the scapula and are inserted into its ventral

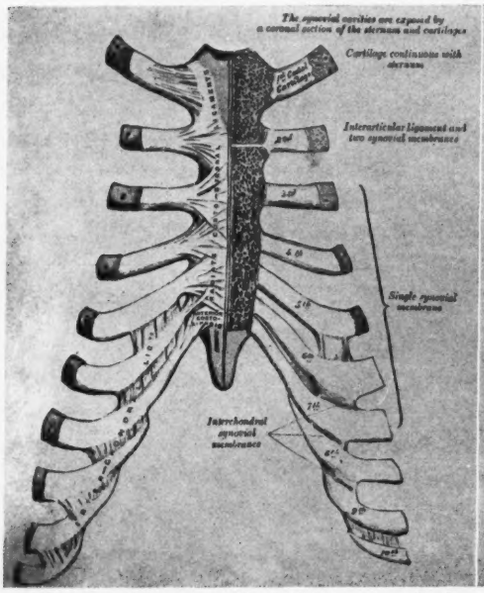
Cartilage continuous with sternum
Interarticular ligament and two synovial membranes
Single synovial membrane
Interchondral synovial membranes

*Fig. 1. Sternocostal and interchondral articulations, anterior view.[10]

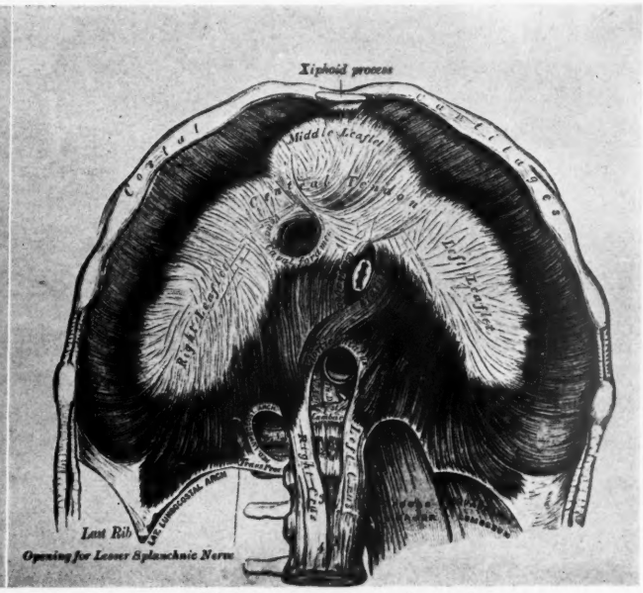

Fig. 2. The diaphragm; undersurface.[10]

* All illustrations are taken from Gray's "Anatomy," Twenty-third Edition. Edited by Warren H. Lewis, B.S., M.D., Philadelphia and New York, 1936. Lea and Febiger.

surface. The serratus anterior functions as a whole to carry the scapula forward, and at the same time raises the vertebral border climbing, and raise the lower ribs in forced inspiration. Its nerve supply is the long thoracic.

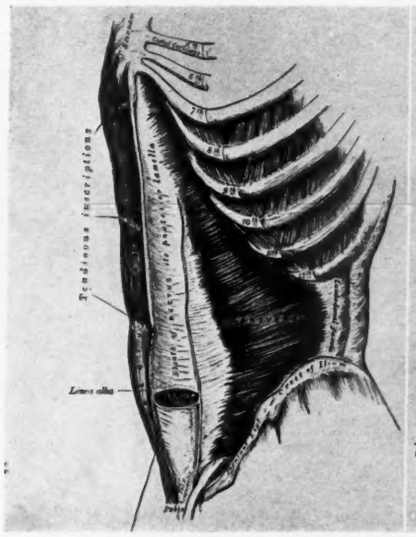

Fig. 3. The transversus abdominis.[10]

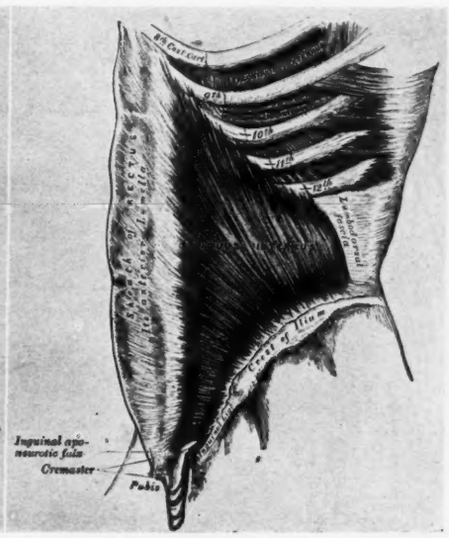

Fig. 4. The obliquus internus abdominis.[10]

of the bone. It is therefore concerned in the action of pushing. It stabilizes the scapula and assists the trapezius muscle in motions at the sternoclavicular joint, the deltoideus muscle in raising the arm, and may assist in raising and everting the ribs. The nerve supply is "by the long thoracic, which is derived from the fifth, sixth and seventh cervicals," by way of the brachial plexus.

The latissimus dorsi (Fig. 5) is a triangular, flat muscle, which covers the lumbar region and lower half of the thoracic region. It arises from the spinous processes of the lower six thoracic vertebrae, the lumbodorsal facia, the supraspinal ligaments and the external lip of the crest of the ilium. It also arises from the three or four lower ribs, interdigitating with the processes of the external oblique muscle. This muscle passes obliquely upward, crosses the inferior angle of the scapula, to which it is lightly attached, and converges into a tendon which is inserted into the upper anterior aspect of the humerus below the lesser tuberosity. Its motion is concerned with the downward pull of the arms, as in felling a tree and in sabre practise. It may assist the pectoral or abdominal muscles in bringing the trunk forward, as in

Posteriorly attached to the lower ribs, coming from above and below, are the deep muscles of the back, including the iliocostalis lumborum and the dorsi group, notably the longissimus dorsi. Their action is to assist in bending and stabilizing the spinal column and the trunk. Their nerve supply is the posterior primary divisions of the spinal nerves, closely associated with the sympathetics.

I have mentioned the muscles directly attached to the eighth, ninth and tenth ribs and their cartilages, but many other muscles and nerves are involved in the intricate mechanical processes of motion of the rib borders. The above is sufficient to suggest an explanation for the many signs and symptoms that characterize the syndrome of slipping rib cartilage.

ETIOLOGY

Abnormal mobility of the anterior ends of these anterior rib cartilages may begin acutely as a partial separation of the fibrous attachment. On the other hand, it may be the result of multiple injuries—stretching of the fibrous attachments over a period of time, as from golfing or one-sided weight carrying.

That trauma or injury, direct or indirect, is the etiological background seems reasonable. Ballan and Spector[13] report eight

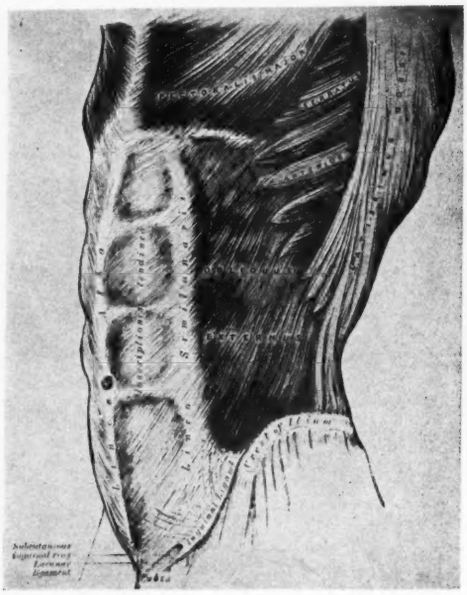

Fig. 5. The obliquus externus abdominis muscle, also indicating the serratus anterior and the latissimus dorsi.[10]

cases, under the title "Slipping Rib" and state in summary, "In most instances the slipping rib develops as a result of injury but that the patient frequently fails to attach any importance to the injury."

Deformity of the loosened rib cartilage end may result from displacement of a fracture, as appeared in one case; but usually it is due to the curling upward of the loosened cartilage end, so that the curled end rises above and to the inside of the superior rib cartilage, mechanically slipping up and down on respiration. It also slips out and in over the rib border on certain motions of the chest and arms, and on digital manipulation, with a click and a pain which is diagnostic.

From analyzing sixty-eight cases, fifteen reported prior to 1938,* seven by personal communication and forty-six of my own, it appears that slipping rib cartilage results more often from indirect than from direct trauma, there being thirty-six of the first and fourteen of the latter. In six cases, both direct and indirect force was in evidence. Of the twelve other cases no attempt was made to establish a cause due, I believe, to incomplete histories. It is usually necessary to retake the histories of these cases; for, since the cartilage deformity develops over a period of time subsequent to the trauma, the patient frequently does not associate the injury with the complaint and the cause is not recognized. This is particularly true of the cases of indirect injury.

An example of direct injury is the sudden application of a steering wheel against the lower ribs. Indirectly it may be caused by sudden flexion, extension, or twisting of the body, by repeated arm pulls, as in golfing, by a sudden pull on the arms in any form of lifting, by pushing, by the act of forced expulsion, as childbirth or coughing, and by many other types of force, any of which may cause or aggravate the condition.

In explaining the pain and tenderness associated with this syndrome, Cyriax[1] said, "Pain and tenderness produced by displacement of the anterior ends of the ribs or cartilages is doubtless due to irritation of the intercostal nerves in the vicinity, from which it may radiate to the posterior spinal nerves and thence to the thoracic or abdominal sympathetics." A diagram of the course and branches of a typical intercostal nerve, from Gray's[10] *Anatomy*, page 945, Figure 819, shows the logic of this statement.

According to Gray,[10] the intercostal nerves "pass forward in the intercostal spaces below the intercostal vessels. At the back of the chest they lie between the pleura and the posterior intercostal membranes, but soon pierce the latter and run between the two planes of intercostal muscles as far as the middle of the rib. They then enter the substance of the intercostals interni, and, running amidst their fibers as far as the costal cartilages, they gain the inner surfaces of the muscles and lie between them and the pleura. Near the sternum, they cross in front of the internal mammary artery and transversus thoracis muscle, pierce the intercostals interni, the

* The eight cases of Ballan and Spector are not included in this analysis.

anterior intercostal membranes and pectoralis major, and supply the integument of the front of the thorax and over the mammae, forming the anterior cutaneous

tribution and communication of the thoracicoabdominal intercostal nerves may further explain the widespread and varying pain symptoms.

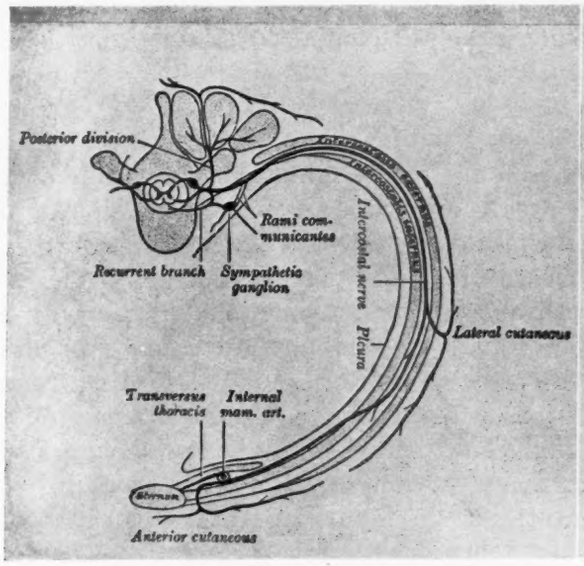

Fig. 6. Diagram of the course and branches of a typical intercostal nerve.[10]

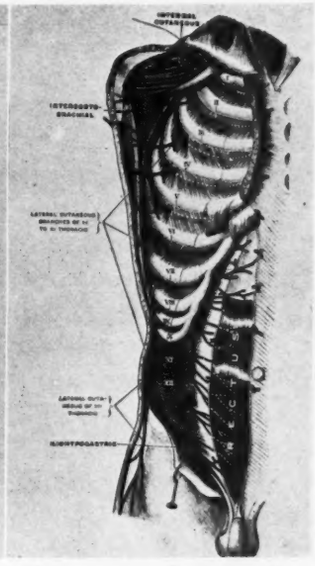

Fig. 7. Intercostal nerves, the superficial muscles having been removed (testus).[10]

branches of the thorax. . . . Each nerve is connected with the adjoining ganglion of the sympathetic trunk by a gray and white ramus communicans."

Another diagram from Gray's[10] Anatomy, page 947, Figure 221, further depicts the course and distribution of the thoracicoabdominal intercostal nerves (the seventh to the eleventh inclusive), their communication with the iliohypogastric nerve of the lumbar plexus, and particularly shows the close relationship at the costochondral junction of the vertebrochondral ribs, so that if the end of the vertebrochondral anterior rib cartilage becomes loosened, deformed and slips up under the rib cartilage above, there is a strong probability of nerve irritation. Substantiating this reasoning is the evidence that when the loosened and deformed cartilage is removed, the pain disappears immediately and permanently.

Keeping this diagram in mind, one can understand that the nerve above the lesion is particularly involved, and that the dis-

As pointed out by Cyriax,[1] and later by Davies-Colley,[2] the association of the sympathetic system with the intercostal nerves accounts for pain symptoms which suggest deeper lesions of the abdomen and thorax. Whether the interchondral joints which are involved in slipping rib cartilage contribute in any way to the pain manifestation is worthy of consideration.

Pathological examination of specimens obtained from these operations has shown nothing of interest.

Deformity of the loosened cartilage to produce the click usually develops secondarily, and therefore may not be recognized as coming from the original injury.

With these points in mind, I believe that a carefully taken history will, in all instances, show the etiology to be direct or indirect trauma.

DIAGNOSIS

Diagnosis of slipping rib cartilage is made from the history of pain in the chest or abdomen, usually, in part at least, at or

near the rib border, and over a period of time. Some of the patients complain of a slipping sensation or of something giving away at the rib borders, associated with pain. Others speak of bunches on, or of soreness of, the rib borders. By digital examination with the patient in the supine position and the knees flexed, an area of tenderness at the rib border is noted, especially when the examiner's fingers are well under the rib border and pressing outward. At the same time the abnormally movable rib cartilage, with its associated click and pain, may be demonstrated. The slipping rib cartilage is easy to demonstrate in some cases and difficult in others so that, given a suggestive history, and an area of tenderness at the rib border, a tentative diagnosis can be established and repeated examinations or examination under anesthesia made to confirm it. This is especially true of the acute case in which muscle spasm is likely to prevent satisfactory local examination.

The intensity of pain complained of is frequently well away from the rib border in the anterior chest wall, the breasts, the heart region, the shoulder blades, the back and the abdomen, but usually there is an associated general soreness of the "ribs" and an acute localized tenderness at the rib border. It is important to rule out other possible causes or factors, especially when the pain symptoms involve the abdomen and raise the question of an intra-abdominal lesion, and here *x*-ray examination is of great assistance. The same is true of the chest when a question of fractured rib is considered, either as a cause or contributory factor of pain.

Limitation of chest expansion as demonstrated by measurement is a suggestive diagnostic feature.[12]

Positions of carriage and action of the body and limbs are noteworthy. Some patients are bent forward and to the affected side, some cannot raise their arms without causing pain. Some have pain in bending forward and rising from a forwardly bent position, so that they accomplish this act by crouching and rising with the back straight.

It is of the greatest importance in diagnosis to have this syndrome in mind and also to have a clear understanding of its development.

TREATMENT

Cyriax[1] treated his patients conservatively, but other cases reported have been treated mainly by excision of the loosened cartilage. This has usually resulted in immediate and permanent relief of symptoms.

Personally, I have treated the acute condition conservatively by adhesive strapping. Later, in the course of one to three months or longer, if the symptoms persist, and with sufficient severity, I advise operation, excision of the loosened rib cartilage.

Some of my patients have, in part at least, recovered under conservative treatment; some of them have declined operation, preferring to tolerate the pain or are still considering operation. Those patients, twenty-two in number, whose symptoms had continued and who submitted to operation, were treated by excision of the rib cartilage or cartilages involved with excellent and often dramatic results.

OPERATION

The incision is made in the direction of the slope of the ribs, three finger-breadths above the umbilicus and centered at the anterior-axillary line; or starting at a point one finger-breadth anteriorly and above the tip of the eleventh rib cartilage (which can easily be felt) the incision is made in the direction of the slope of the ribs to the mid-axillary line. Having exposed the muscles, the operator's fingers are hooked under the rib border, and an examination made of the tenth, ninth and eighth rib cartilages, identifying the cartilage or cartilages involved. The surgeon supports the loosened cartilage with the fingers and the muscles are separated down to the cartilage which is to be removed and back to its articulation with the rib. After pushing the anterior muscle attachments away disarticulation is

performed, using a scalpel. The disarticulated end of the cartilage is grasped in double hooks, the muscle attachments, laterally and beneath, are dissected off and the cartilage removed. A further examination is then made of the adjacent cartilages for any abnormal notion or deformity. If found, these cartilages should also be removed. If not found, the incision is closed. Beginning at a point between the end of the operated rib and the rib above, a suture of plain catgut is taken through the intercostal muscles. To this point the adjacent intercostal muscles, previously detached from the cartilage, are drawn in for protection. Continuing with the same suture the external muscle rent is brought together and the skin incision closed.

CONVALESCENCE

Convalescence is usually uneventful. Temporary bowel and urinary inaction may be expected as the operation involves muscles that assist in these expulsive acts. The patient is immediately relieved of the "old pain" and leaves the hospital in from three to ten days. It is important to discriminate between the "old pain" and the pains that patients sometimes have following this, or any other type of operation; hence the value of a carefully taken preliminary history. Otherwise the immediate operative results may be wrongly interpreted.

ANALYSIS OF CASES IN WHICH OPERATION WAS PERFORMED

Of thirty-seven patients operated on, eight in the literature prior to 1938, eight by personal communication and twenty-one of my own, the ages ranged from six to fifty-seven years; the condition occurred on the left in fourteen cases, on the right in thirteen cases, and bilaterally in ten cases.

One rib was involved in twenty-one cases, two ribs in 11 cases, three ribs in one case and four ribs in four cases.

There was an area of tenderness at the site of the lesion in all cases. There was a wide variation in the character of pain. In some cases it was dull, in others sharp, especially on manipulation of the cartilage; in still others it was gripping or pulling. In some cases the pain was so modified that it was hardly recognized as such by the patients until questioned, and they frequently did not realize their handicap until they had been relieved of the annoyance.

Pain was constant in some cases; in some it occurred with different positions of the body or after certain types of muscle pull. Instances occurred in which remission of symptoms was brought about by rest from work or from forced rest in bed, occasioned by illness.

Following excision of the offending cartilage or cartilages, cure was obtained, permanent and usually immediate. There was no mortality.

CASE REPORTS

Case 1. F. T., a seventeen-year-old shoeworker, was first seen at my office on October 28, 1938. He stated that two days previously he caught his left sleeve in a roughing brush and was pulled up to the machine with a twisting motion, lacerating his upper arm. At that time, aside from the arm injury, he felt a sharp pain in the left rib border, and had noticed some soreness there since.

Physical examination revealed an abrasion and contusion of the left upper arm with infection, localized tenderness at the left rib border, and general soreness of the left anterior chest.

The arm lesion healed readily, but the chest symptoms persisted. One month later, an examination showed tenderness still present at the left rib border, and, on digital manipulation (the patient in supine position, with the knees flexed) the anterior end of the tenth anterior rib cartilage was found to be abnormally loosened and deformed—curled up under the rib cartilage above. It could be slipped out over the rib border, producing a click and a pain. The diagnosis was slipping rib cartilage of the tenth left rib. The type of force to produce this injury was indirect, from pull on the arm and torsion of the body at the time of the accident.

Treatment was conservative at first, the chest being strapped with adhesive plaster.

The condition grew worse, and the patient became incapacitated for work. On September 10, 1940, at the Sacred Heart Hospital, Manchester, New Hampshire, an operation was performed. The anterior end of the tenth anterior rib cartilage was found to be abnormally loosened and curled up under the rib cartilage above. On manipulation, it could be brought out over the rib border, producing a click. The loosened and deformed cartilage was excised. The patient was immediately and completely relieved of symptoms and left the hospital in six days. His convalescence was short and uneventful. He returned to work in about four weeks, has remained well and has worked steadily.

Case II. A. F., a fifty-one year-old W.P.A. sewing project worker, was first seen at my office on January 10, 1940. She stated that on that day she caught her toe in the handle of a bulkhead door and fell on a concrete sidewalk with the left arm under her. At the same time she injured both knees. She complained of pain in, and general soreness of, the anterior chest with especial tenderness on the left side along the rib border, also of pain in the left upper arm and both knees.

Physical examination revealed general soreness of the anterior chest, particularly on the left side and more especially at the left rib border, contusion and discoloration of the left upper arm, laceration and swelling of both knees. The arm and knee injuries gradually healed but the chest symptoms persisted.

On April 11, 1940, her complaint was pain in the left rib border, aggravated by stooping, by lateral motion, by deep breathing, and by raising of the left arm. Examination showed localized tenderness at the left rib border, with a deformity and slipping of the anterior end of the ninth left anterior rib cartilage, associated with pain. The diagnosis was slipping rib cartilage of the ninth left rib. The type of force to produce this injury was direct, from pressure of the left elbow on the rib border at the time of the fall.

The treatment at first was conservative, consisting of adhesive plaster strapping of the left chest, but no relief was obtained. On April 23, 1940 (three months after the injury), an operation was performed at the Elliot Hospital, Manchester, New Hampshire. The anterior end of the ninth anterior rib cartilage was found to be abnormally loosened and deformed—curled up under the rib cartilage above. On manipulation it came out over the rib border with a click. The ninth left anterior rib cartilage was excised. Disappearance of pain was indefinite at first, and convalescence was slow. On May 3, 1940, the patient was discharged from the hospital, relieved; but she did not return to her employment until about three months later. Since then she has worked steadily.

Case III. M. A. F., a thirty-year-old domestic, was first seen at my office on February 23, 1940, at the request of Dr. Roland Joyce of Nashua, New Hampshire. She stated that on December 30, 1939, while riding in an automobile which was involved in an accident, she suffered a direct injury to her chest. She felt some pain, and the following day noted discoloration at the right rib border; but she considered her condition casual and did not see her physician until three weeks later, when she complained of pain at the rib borders, exaggerated by deep breathing or by twisting and flexing of the body. The symptoms became more severe until she was incapacitated for work.

Physical examination revealed a localized tenderness at the rib borders, with slipping and deformity of the anterior end of the tenth anterior rib cartilage, bilaterally. The diagnosis was slipping rib cartilage of the tenth rib, bilaterally. The type of force to produce this injury was direct, received at the time of the accident.

On April 8, 1940, an operation was performed at St. Joseph's Hospital, Nashua, New Hampshire. Examination at that time showed the anterior end of the tenth anterior rib cartilage on both sides to be abnormally loosened and deformed—curled up under the corresponding rib borders. On manipulation they could be brought out over the rib cartilages above with an audible click. The eleventh ribs and their anterior cartilages were abnormally long. The tenth and eleventh anterior rib cartilages on either side were excised, the eleventh being removed for symmetry in relation to the anterior ends of the tenth ribs. The patient made an early and complete recovery. On May 19, 1941, Dr. Joyce reported that she remained completely relieved and had worked steadily.

Case IV. M. L., a twenty-year-old shoeworker, was first seen at my office on August 16, 1940. She stated that following childbirth fourteen months previously, she began to have pain in her right side. (In 1937 she had been operated

upon for gallbladder disease and the gallbladder removed, following which she remained well for two years.) After childbirth, when she began to experience pain in the gallbladder region, she felt that her symptoms were due to adhesions resulting from the gallbladder operation. The pain, which was definitely localized at the right rib border, became progressively worse, and she had been unable to work for one year.

Physical examination revealed general tenderness about the right upper quadrant of the abdomen and adjacent lower chest, with a localized area of tenderness at the right rib border. There was an abnormal mobility and a deformity of the anterior ends of the ninth and tenth right anterior rib cartilages, which, on manipulation, slipped out over the rib border with a click and a pain—recognized by the patient as her complaint. The diagnosis was slipping rib cartilage of the ninth and tenth right ribs. The type of force to produce this injury was indirect, from muscle pull in stabilization of the lower chest, and abdominal compression in the expulsive act of childbirth.

On August 27, 1940 (fourteen months after the injury), an operation was performed at the Elliot Hospital, Manchester, New Hampshire. The abnormal looseness and deformity of the anterior ends of the ninth and tenth right anterior rib cartilages were demonstrated and the two cartilages removed. The patient was immediately and completely relieved of symptoms and was discharged from the hospital in five days. She returned to her work one month latter and has remained well ever since.

Case v. C. S., a forty-year-old cloth inspector, was first seen at my office on September 14, 1940, at the request of Dr. Joseph A. Sapienza of Lawrence, Massachusetts. The patient stated that on September 1, 1940, while lifting a heavy bolt of cloth from the floor, he felt a sharp pain in his right chest near the rib border. The pain had continued and was aggravated by deep breathing, stretching, coughing, bending of the body, or by any motion requiring the use of the chest muscles on the right side. The symptoms had become progressively worse and he was incapacitated.

Physical examination revealed that the pain point, of which he complained, was about two inches above the right rib border. The tenderness, however, was at the rib border, and the anterior ends of the ninth and tenth right anterior rib cartilages were found to be abnormally loosened and deformed—curled up under the rib cartilages above. On manipulation, which was difficult because of acute muscle spasm, a click, with an associated pain, was demonstrated. The diagnosis was slipping rib cartilage of the ninth and tenth ribs. The type of force to produce this injury was indirect, from muscle pull in chest stabilization and abdominal compression during the act of lifting.

On September 23, 1941, at the Clover Hill Hospital, Lawrence, Massachusetts, an operation was performed. The abnormally loosened and deformed ninth and tenth right anterior rib cartilages were demonstrated and removed. The patient was immediately relieved, made an uneventful recovery and returned to work in about three weeks. On May 16, 1941, Dr. Sapienza reported him to be free of symptoms and in excellent health.

Case vi. L. St. A., a twenty-two-year-old mill hand and laborer, was first seen at my office on March 25, 1941, at the request of Dr. R. C. Seed of Lawrence, Massachusetts. The patient stated that on January 16, 1940, while pushing a heavy truck, he felt a pain in his rib borders, especially on the left side. As he continued to work, the pain became worse from day to day. He felt it most severely while working, but was uncomfortable for some time after he stopped working. The symptoms became intensified, and after two weeks he was given a lighter job, at which he continued for eleven months. His complaint was pain at the rib borders, particularly on the left, while standing erect, while walking about, in kneeling, and in other motions of the body. He was incapacitated for work.

Physical examination revealed a localized tenderness at the rib borders, with an abnormal mobility and a deformity of the anterior ends of the ninth and tenth anterior rib cartilages on either side. By digital manipulation, these curled up cartilages could be brought out over the corresponding rib borders, producing an audible click, with an associated pain. The diagnosis was slipping rib cartilage of the ninth and tenth ribs, bilaterally. The type of force to produce this injury was indirect, from muscle pull in the act of pushing, and over a period of time.

On June 10, 1941, at the Clover Hill Hospital, Lawrence, Massachusetts, an operation was performed. The abnormally loosened and deformed ninth and tenth anterior rib cartilages on either side were demonstrated and removed.

On June 28, 1941, Dr. Seed reported that the result was very satisfactory; there had been no complaint of the old pain since the operation.

Case VII. P. St. G., a six-year-old school boy, was first seen at my office on March 31, 1941. His mother stated that in August, 1940, she was riding in the back of a truck, sitting on an improvised seat and holding the child on her lap, when a collision with an automobile occurred. When she saw the imminence of the collision, she grasped the child tightly about the lower chest. Both were thrown forward, and the child came in contact with another board seat in front, bruising his left rib border. He was taken to a hospital, *x*-rayed, and examined by a physician. Subsequently, he was re-examined by another physician and *x*-rayed at another hospital, with negative reports. Meanwhile he had complained of pain at the rib borders, particularly on the left side. He cried, walked with his body bent forward, and could not play like other children, because of pain at the rib borders.

Physical examination showed his general condition to be good. There was tenderness at the rib borders, and on digital manipulation the anterior end of the tenth anterior rib cartilage on either side was found to be abnormally loosened and deformed—curled up under the corresponding rib cartilage above, and when brought out over the rib border, produced a click, with an associated pain. The diagnosis was slipping rib cartilage of the tenth rib, bilaterally. The type of force to produce this injury was direct, from pressure of the mother's arms, and from contact of the chest with the seat in front at the time of the accident.

On June 12, 1941, at the Hillsborough General Hospital, Grasmere, New Hampshire, an operation was performed. The abnormally loosened and deformed tenth anterior rib cartilage, on each side, was demonstrated and removed. The patient was immediately relieved of his symptoms and was discharged from the hospital on June 24, 1941. There has been no complaint since and the child walks and plays in his natural way.

Case VIII. B. B., a seven-year-old school boy, was first seen at my office on May 5, 1941. His complaint was of intermittent pain across the upper abdomen. His mother stated that he had a severe attack of whooping cough in April and May of 1940. The pain attacks in the upper abdomen began in July, 1940. They became worse, and the mother thought they might be due to appendicitis. Recently the child was examined at a clinic where the mother was told that he did not have appendicitis. He continued to have abdominal pains, and his mother then wondered if they might be due to adhesions from a hernia operation performed two years previously. During the two weeks just passed, the pain attacks had been more severe and more frequent, occurring every day, and on the day before he had suffered six attacks.

Physical examination showed the patient to be fairly well developed and nourished. He walked with a stoop and held himself rather tense. There was localized tenderness at both rib borders, and the anterior end of the tenth anterior rib cartilage on either side was found to be abnormally loosened and deformed—curled up under the corresponding rib cartilage above, and on digital manipulation could be brought out over the rib border, producing a click with an associated pain which corresponded to the symptoms. The abdomen was negative. The previous hernia repair was in excellent condition with no evidence of recurrence. The diagnosis was slipping rib cartilage of the tenth rib, bilaterally. The type of force to produce this injury was indirect, from the muscle pull in coughing, which began in April and May of 1940, during the attack of whooping cough; but the deformity to produce the pain developed later so that the etiological factor in the case was obscure.

On May 6, 1941, at the Elliot Hospital, Manchester, New Hampshire, an operation was performed. The loosened and deformed cartilages as previously described, were demonstrated and removed. On May 8, the patient stated that the pain was gone, and on May 12, six days after the operation, he was discharged from the hospital. He has remained entirely free from symptoms and walks in a normal way.

SUMMARY AND CONCLUSIONS

1. Slipping rib cartilage is a definite lesion involving the anterior ends of the anterior cartilages of the vertebrochondral ribs, namely, the eighth, ninth and tenth on either side. It is not concerned with the costochondral articulations.

It is due to a loosening of the fibrous hammock-like attachment of the anterior end of the cartilage, and the loosening is

followed by deformity—a curling upward of the cartilage end—so that it rises to the inside of the rib above and comes in close relation to the intercostal nerve which is the seat of pain.

It is always of traumatic origin, either direct or indirect, more often the latter.

It occurs singly, and as multiple and bilateral lesions.

Age or sex are of no consideration.

2. The cartilage deformity develops over a period of time subsequent to the injury. The patient frequently does not associate the injury with the complaint, and the cause is not recognized; this is particularly true of indirect injury.

Since the rib border is the weakest part of the chest wall, a direct injury, even of minor severity, may cause the lesion, especially in the nonathletic.

Due to the muscle attachments and the different directions and degree of muscle pull, this loosening may occur in many ways.

There is a wide distribution of nerves involved, namely, the intercostals connected with the sympathetic system, and the brachial and lumbar plexuses. By way of the sympathetics, the intercostal nerves are connected with the cardiac, the solar or epigastric, and the hypogastric plexuses which, in turn, have branches to the viscera.

Thus the pain manifestations cover a wide field. The intensity of pain complained of is frequently well away from the rib border—in the anterior chest wall, breast region, shoulder blades, back, abdomen, and so forth; but usually there is an associated general soreness of the "ribs" (so spoken of by the patient), and a localized area of tenderness at the rib border.

3. Diagnosis is made from the history of pain in the chest or abdomen over a period of time. Usually the pain is in the anterior chest, at or near the rib borders.

There is a localized area of pain at the rib border, the site of the lesion.

By digital manipulation, with the patient in the supine position and the knees flexed, the abnormally loosened and deformed cartilage can be brought out over the rib border with a click and a pain that is diagnostic.

X-ray examination is of assistance in ruling out deeper lesions of the chest and abdomen.

4. The pain of slipping rib cartilage is not like other pains. It is usually a dull ache and is often tolerated for years, even a life time. Some patients scarcely realize that they are impaired until operation is performed and their annoyance is taken away. Others suffer severely, and are acutely and completely incapacitated.

There are many cases of obscure pain associated with the chest and abdomen which may have as their background the slipping rib cartilage. Therefore, examination of the rib borders should be made routinely, and information relative to the syndrome should be generally disseminated.

The acute condition should be treated conservatively. Later, in the course of from one to three months, if the symptoms persist and with sufficient severity, excision of the loosened, deformed cartilage or catilages involved should be advised. Operation results in permanent and usually immediate relief of symptoms. There has been no mortality.

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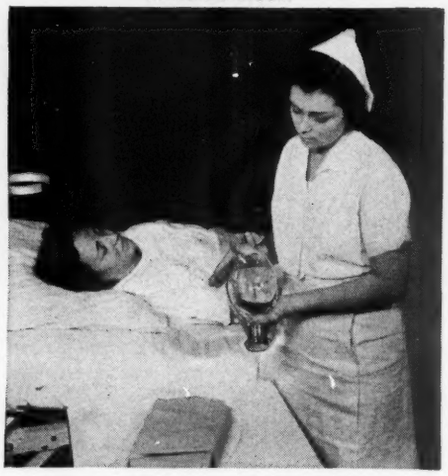

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Hanover, N. H., September 5–6, 1941. (Secretary—DR. JOHN F. GILE, Hanover, New Hampshire)

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Los Angeles, February 19–22, 1941. (Secretary—DR. H. GLENN BELL, University of California Hospital, San Francisco)

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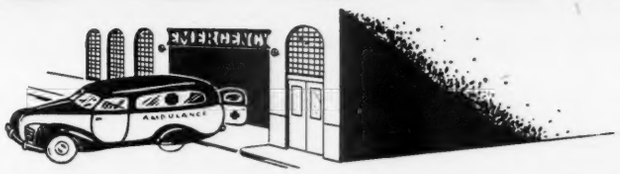

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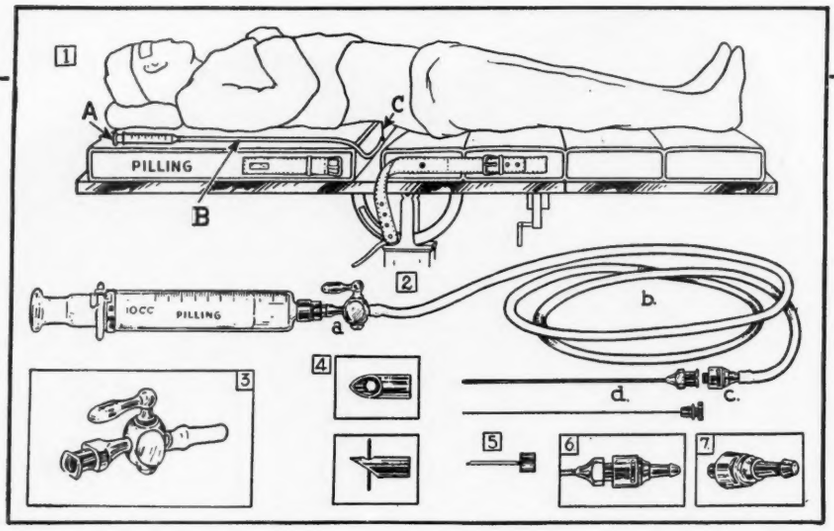

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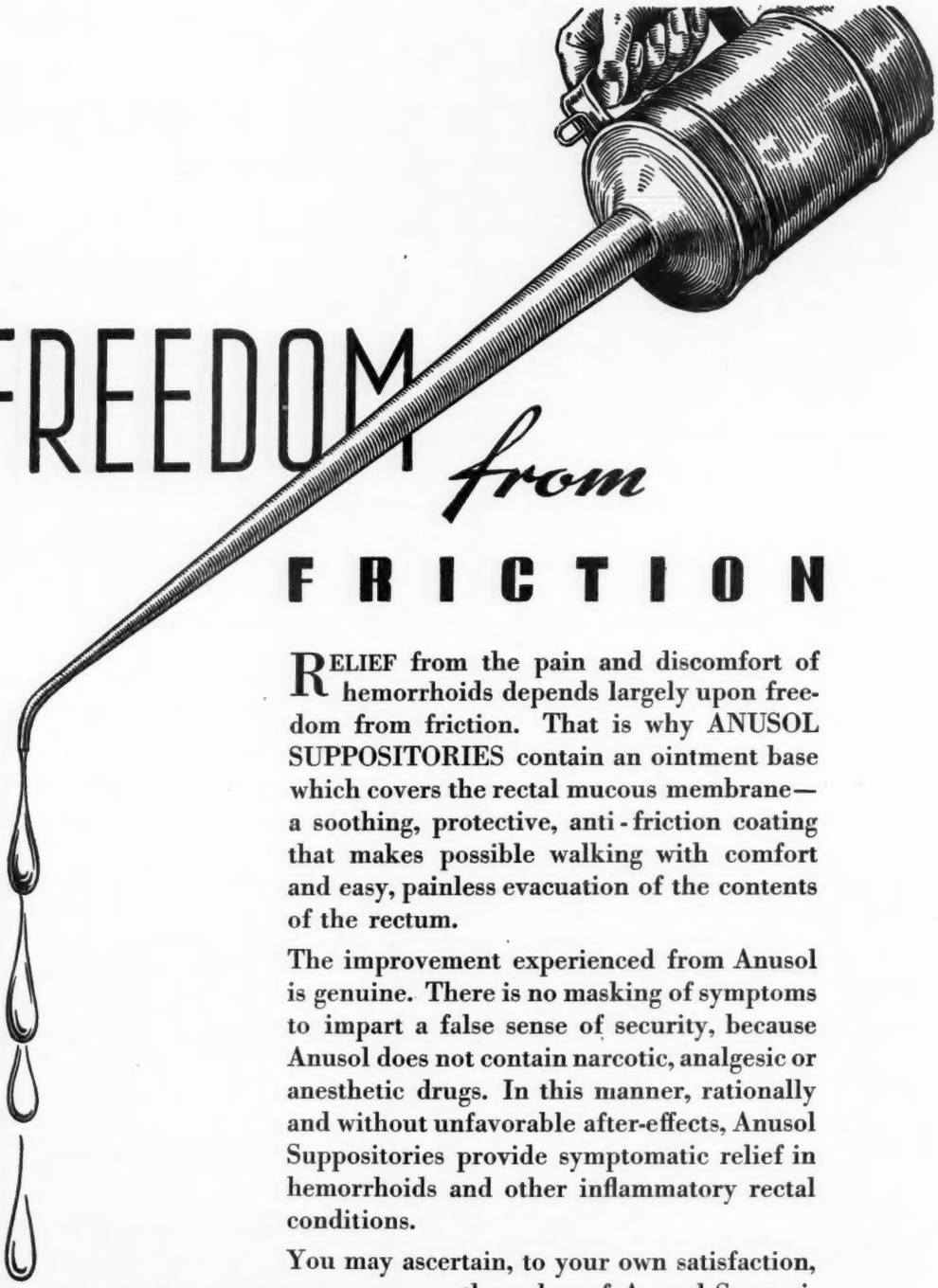

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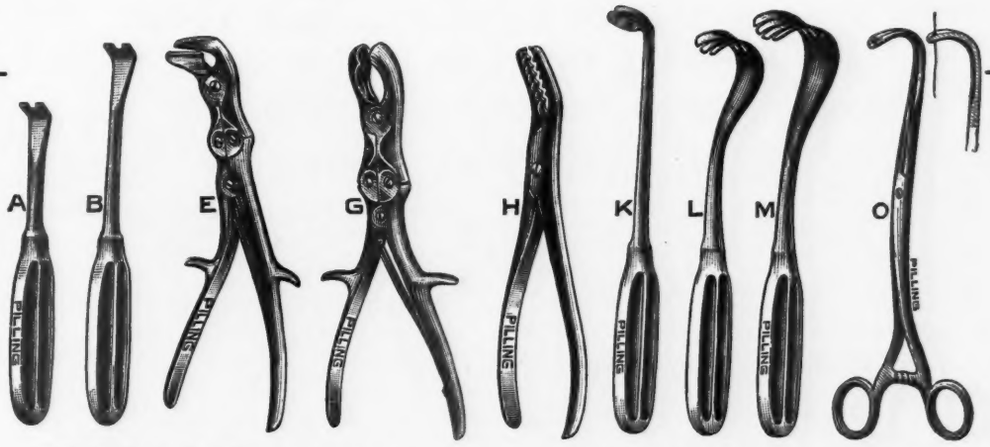

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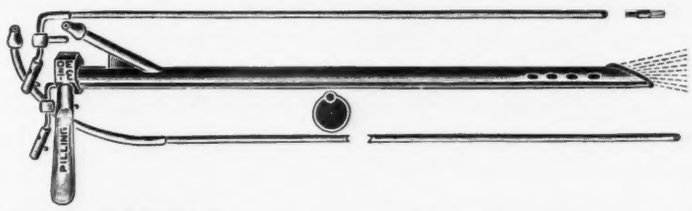

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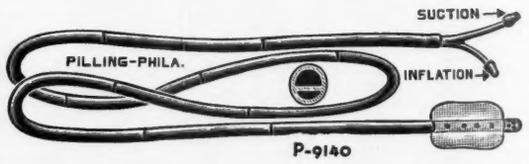

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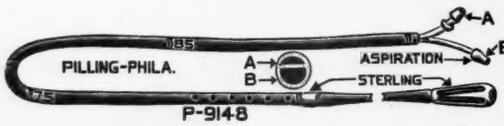

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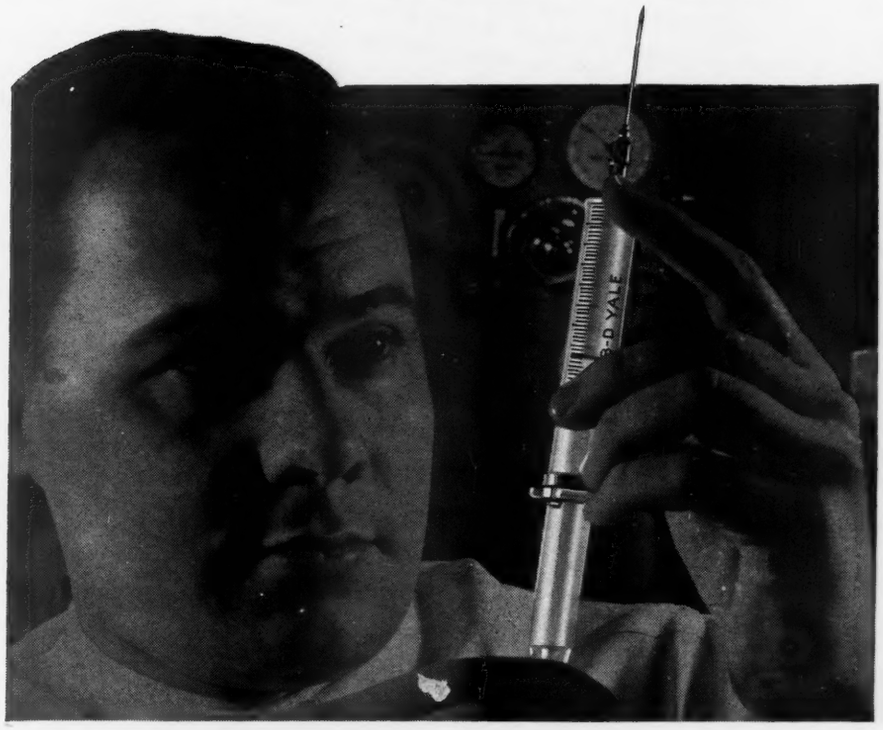

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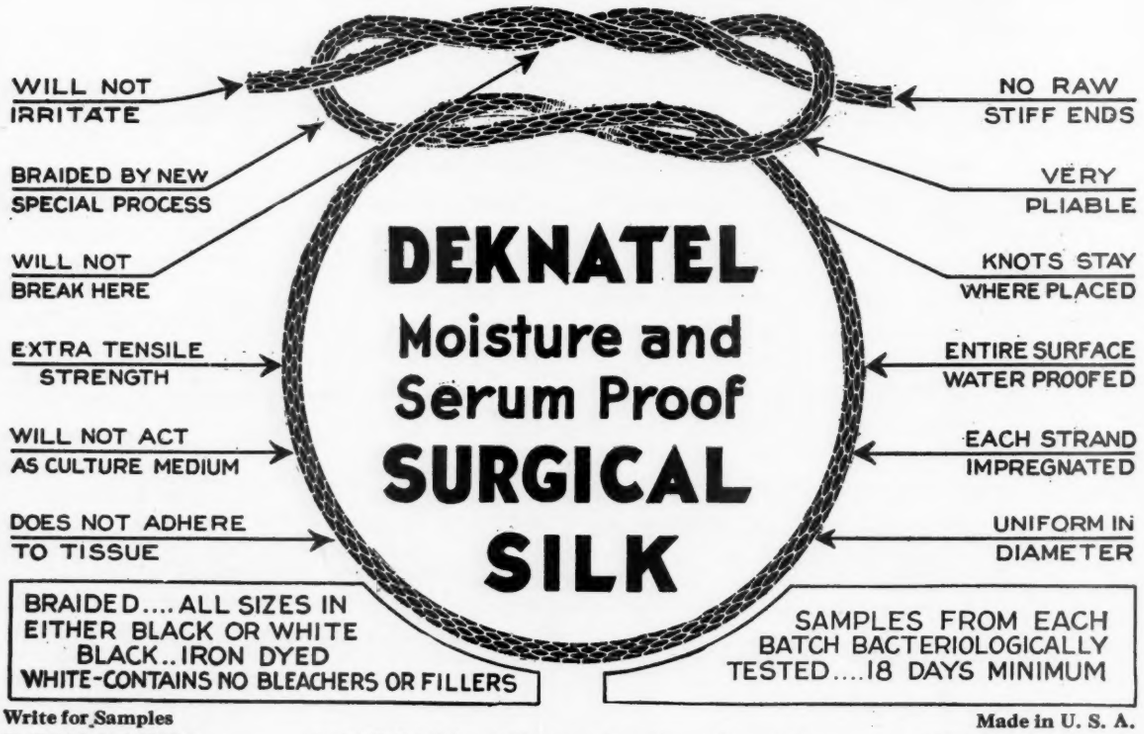
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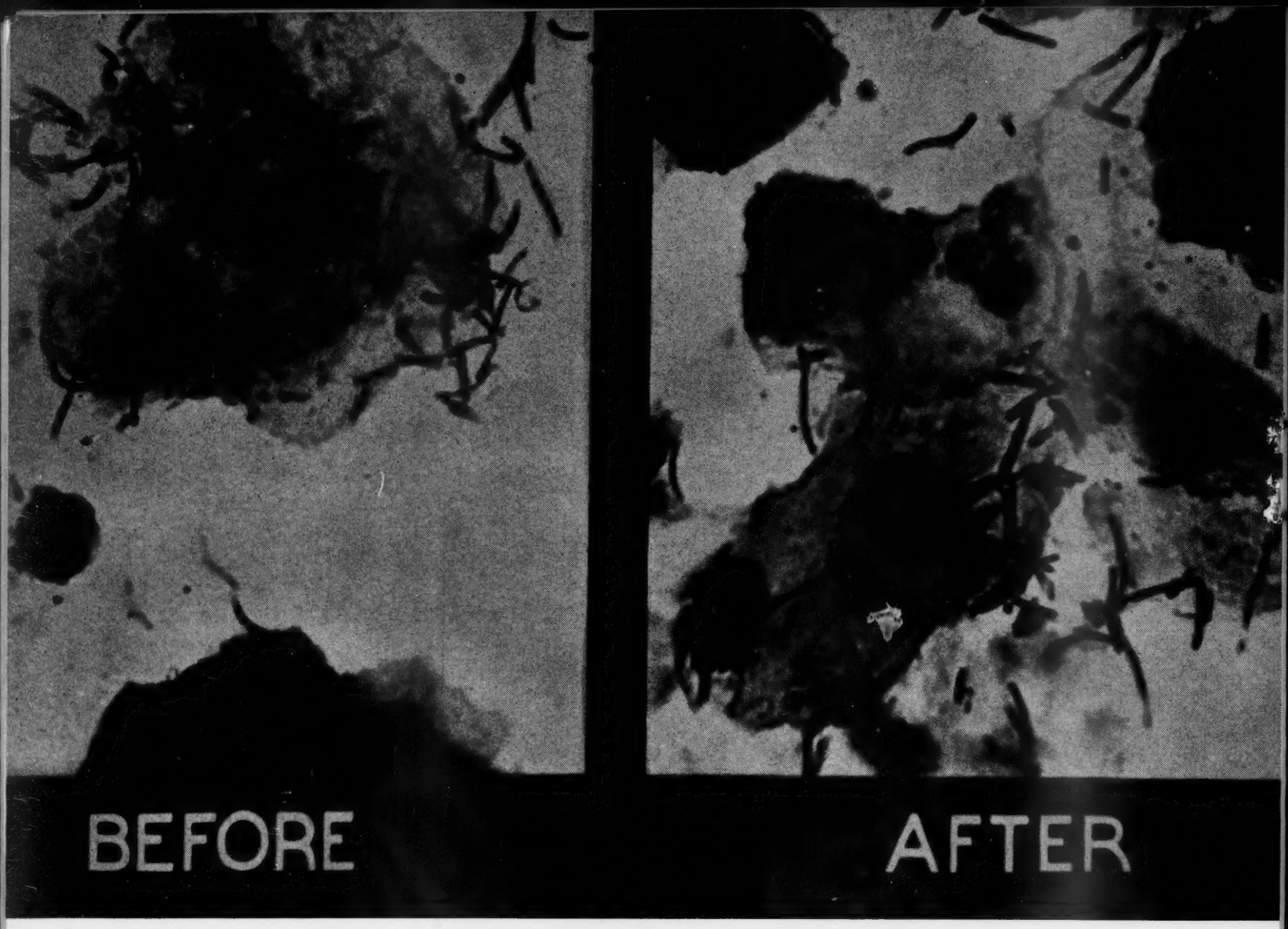

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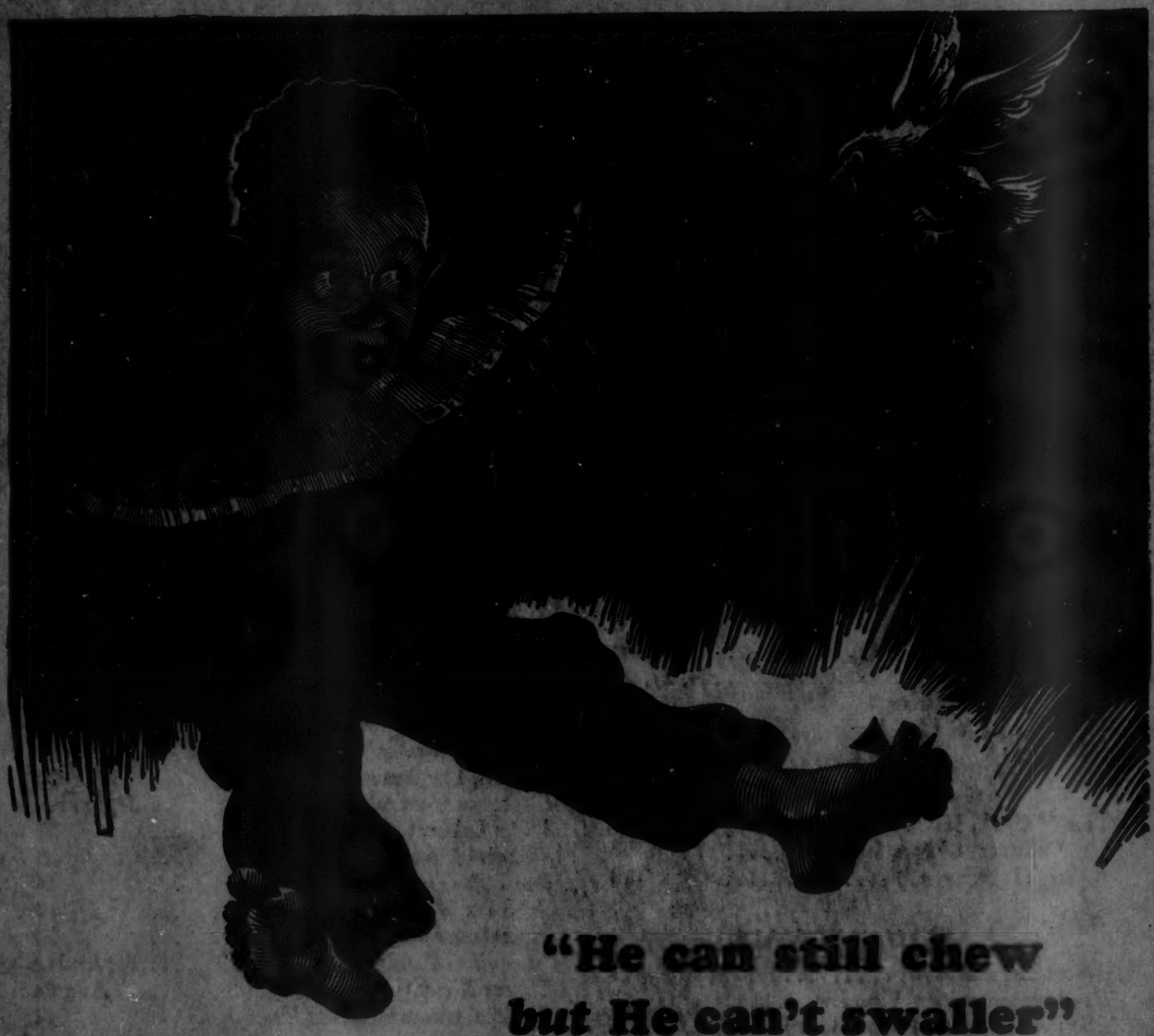

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